"FOOT ROT OF CEREALS"

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CONTENTS.

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INTRODUCTION.

The problem of 'Foot Rot' and 'Root Rot' of cereals has been under consideration for the last few years in almost all the cereal growing countries. Most of the work in this direction however, has been carried out in U.S.A. and Canada and much of our knowledge is due to the workers in these two countries, although important contributions on the subject have also been received from various European countries including Britain. These two diseases are almost invariably accompanied by certain allied diseases commonly known as the 'Seedling Blight', 'Thinning Out','Take All', 'Spring Yellows', 'Stem Rot', 'Crown Rot', 'White Heads', 'Deaf Ears' and so on, but their actual relationship was not known until recently. All these diseases were considered to be separate and different organisms were supposed to be responsible for each. Certain names of these diseases are merely synonyms of each other, and all such diseases need not necessarily be found in the same field. Much of this ground has been cleared by certain recent works on the genera Fusarium and Helminthosporium and considerable light has been thrown on the actual relationship of the various diseases mentioned above. Some of these diseases occur consecutively and some simultaneously, and in this new light certain collective names such as'Fusariosis' have been suggested as representing these types of diseases. Although /

Although such names are quite useful to a student of Plant Pathology, they appear to be of little or no value to a farmer; more-over such names cannot always correctly be applied in cases where several organisms are concerned collectively. Before such names can be accepted in literature their relative value should carefully be considered.

In certain cases insects are also responsible for similar diseases but the consideration of these pests is beyond the scope of the present work. The relative frequency of the fungal pests has been more fully investigated and their economic importance realised far more than the insect pests which are of rare occurance. Moreover the fungal pests tend to become epidemic and thus fungi remain by far the most important group of organisms which must be dealt with in cereal pathology.

There are various fungi concerned in these diseases but little is known about their relative importance as causal agents. Many of them have perfect stages in their life-history, but the imperfect forms are also considerable in number. Amongst the chief pests of cereals responsible for the diseases under consideration may be mentioned the species of Pythium, Leptosphaera, Mycosphaerella, Calonectria, Gibberella, Ophiobolus, Typhula, Rhizoctonia, Sclerotium, Helminthosporium, and Fusarium. All these are established parasites of cereals in one country or another. Organisms with doubtful pathogenicity such as Alternaria sp. may also be added. From this list it is obvious that we have to deal with /

with a group of organisms with widely varied affinities, and the study of these organisms requires a considerable time before the relative importance of their pathogenicity can be understood. The problem is further complicated by the fact that it is necessary to investigate these fungi for a considerable period before their characters can be determined.

In most of the important forms such as the species of Fusarium and Helminthosporium the phenomenon of variability is very frequently exhibited and these variations are of considerable importance in the taxonomy and the pathogenicity of the organism in question. A parasitic form may change into a non-pathogen and a non-pathogen may prove to be a parasite under different conditions, or after a certain time under the same environmental conditions. This behaviour has puzzled workers for some considerable time and in many cases a serious parasite has been declared harmless simply on the basis that under given conditions the organism failed to infect the host. It may be true that a certain strain of a parasitic form constantly refuses to infect the host under certain conditions but this does not eliminate its importance as a parasite. The organism in question may be in an inactive stage, and its true characters cannot be judged by its behaviour at a certain time. A careful study of the organism must be made before any decision can be given as to its pathogenicity.

Having established the pathogenicity of the fungus the next thing /

thing is to find its identity. Most workers are in the habit of raising their organism to a specific rank after the determination of the genus, simply on the basis that no similar organism has been previously described on the host in question although forms apparently identical have been recorded on related plants. No matter how far the morphological characters may agree the new fungus is atonce raised to a specific rank for the reason mentioned. This system involves another name, and goes a long way to aggravate the already puzzling problem which is thrown still further into chaos rather than simplified.

Careful study of certain species of the various imperfect forms reveals the presence of a number of biotypes within the same species and that each biotype may have several races within its orbit. These races may show remarkable differences at certain times and appear so widely differentiated that the worker is tempted to raise them to a specific rank, although they are merely different races of a certain biotype, let alone species. But it must be borne in mind that there is a considerable variability in all such races and at a certain given phase the race in question may not be exhibiting its normal characters. One phase changes into another and this change may be either permanent or temporary. The temporary changes may be reversible or non-reversible and in the latter case absolutely new forms arise. These new forms are not merely variations within the /

the race as examplified by the temporary reversible changes but are definite mutations. In many cases such changes, along with permanent variations which are also of the nature of mutations, are of considerable importance in genera like Fusarium. A detailed discussion of these changes will be given in the text. Such variations are responsible for certain characters that are shown by an organism and in certain cases are so remarkably different that the existing descriptions of differences between species are no longer of value.

In temporary reversible changes the new characters are exhibited without any definite sequence, and such changes go on playing hide and seek with workers and in fact are even more important than the cases in which the changes are permanent. There seems to be nothing constant in the characters of such organisms and in fact the only constant character appears to be their inconsistancy. Under such conditions the life-histories of these fungi remain a work of speculation and their pathogenicity unestablished. It is impossible to recommend any control measures unless we know all the phases of the life-history of an organism.

Again the geographical distribution of fungi depends mainly on the climatic conditions and the type of crop grown in a certain area. One organism may be the cause of a specific disease in one country, while in another country it may be altogether absent and a different fungus may be the cause /

cause of a similar disease.

These are the varied forms of the problem in hand and the present work is an attempt at an understanding of the various aspects of this problem. Since it is impossible to study all the phases of a certain disease in which more than ten organisms are concerned, the work has been confined to a few aspects of the disease and the life-history of the causal organisms. An attempt, however, has been made to give as much information as possible, especially about the characters of the causal organisms. In the early part of the work the diseases of the oat crop were mainly considered; later on, however, other cereals were included. Some experiments on control measures have also been carried out and promising results have been obtained under green-house conditions. A certain number of fungi have been studied in this connection and accounts of them are given separately in the following pages.

HELMINTHOSPORIUM AVENAE SATIVAE &
H. AVENAE GRAMINEAE. n. vars (sub-sp.).

1- Introductory and Historical.

The present investigation was undertaken with a view to explain the various contradictory statements about the Helminthosporium disease of oats. So far it has been considered that only one species of Helminthosporium, namely H. avenae is parasitic on oats and that other closely allied species like H. gramineum and H. teres do not attack oats, the latter view being confirmed by various workers who failed to infect the oat plant with pure cultures of these fungi. The recent literature on this disease points out two definite sets of symptoms which are so clearly defined by their names, 'Leaf Spot' and 'Leaf Stripe' of oats that there can be no mistake in the rough diagnosis of the disease at first sight. Unfortunately the two have been so confused that they are invariably attributed to the same organism and have been accepted in literature without comment by the most recent workers. Some American authors like Johnson named the stripe fungus H. gramineum as distinct from H. avenae forming leaf spot, but this view was rejected on the basis that H. gramineum obtained from barley did not infect oats and his diagnosis and nomenclature was taken as faulty. Recently in Scotlant O'Brien and Prentice described the leaf stripe of oats and Turner and Millard the leaf spot, but they all attributed it to H. amenae. Their accounts /

accounts of the characters of the fungus are so limited that it is impossible to make out with what they are dealing. From their observations they are justified in diagnosing the organisms concerned as H. avenae, but unfortunately they did not study the fungi closely enough to point out the details which lead to my conclusion that the organisms responsible for the 'Leaf Stripe' and 'Leaf Spot' of oats are different. The difference is so much that the two cannot safely be taken as merely two isolants of the same species but in fact each one can easily be promoted to the rank of a sub-species with several races existing in each. The following brief review of the history also indicates that two distinct diseases are concerned.

Briosi and Cavara in the year 1889 described a Helminthosprium disease of oats in Italy and designated the causal organism as Helminthosporium teres (Sacc). forma avenae sativae differing from the main species in the somewhat smaller dimensions of the spores and existance of conidiophores singly instead of in clusters. Their account of the description of the organism may be given in the following words:- The conidiophores which occur singly scattered are stout, cylindrical, many septate, fuliginous, measuring 150-200 μ X 9-12 μ, and the spores as olivaceous, cylindrical, slightly swollen in the middle and rounded at the tips, 4-6 septate and measuring 80-110 X 15-16 μ. They represent the septa and spore walls as thick structures. The symptoms of /

of the disease as described are the production of narrow, oblong, longitudinally elongated, olivaceous foliar spots with dark margins. The infection starts at the tips of the leaves with the formation of spots surrounded by a pinkish area which gradually melts down to yellow. This yellow area slowly extends down and the leaf is ultimately killed.They do not mention the age of the plants at which the symptoms appear and the early history of the spots on the foliage.

Eidam investigated a leaf spot of oats occuring in Silesia in the year 1891, affecting commonly the first leaf and also the second and third in rare cases. He does not give the description of the spots in definite terms or its actual place of origin. He regarded the organism as a species separate from H. hordei owing to its inability to attack barley and wheat. Later Ritzema Bos described an attack on oats with the production of short, round spots associated with a reddish colour of the leaf and diagnosed the causal organism as H. gramineum.

Ravn in 1901 described a leaf spot of oats as grey or greyish brown lesions accompanied by a reddish colour of the leaves. He does not give definite account of the development of the lesions and describes their shape in the terms of those caused by H.teres on barley, and differentiates them from the latter in having no net-work in the spots and from H.gramineum by the fact that the leaves do not split. He also states that the conidia of this fungus are slightly /

slightly longer than those of H.teres.

Later on certain American workers recorded the presence of H. avenae on straw, but none of them found any lesions on the living plants except Johnson who definitely calls the lesion a stripe and designated the fungus as H. gramineum. Dreschler in the year 1923 published a detailed account of the genus Helminthosporium and gives the symptoms of the disease of oats caused by H. avenae as follows:- Broad and irregular or long and narrow spots with poorly defined margins are found on the leaves and are accompanied by a reddish colour which slowly merges into yellow. Infected leaves wither more quickly and die, the red and orange pigments being replaced by a yellowish grey colour. At this stage the conidia and the conidiophores emerge which closely resemble those of H. teres, although he mentions a greater tendency on the part of the conidiophores to branch. He records the largest number of septa in a spore as nine and its length as 175 μ.

Amongst the British writers, Smith in the year 1923 and again in 1925 records the stripe form of Helminthosporium on oats. O'Brien and Prentice also record the stripe form in Scotland in the year 1931. Turner and Millard describe H. avenae in 1931 as the cause of the leaf spot of oats and stress the importance of the presence of a stripe form which differs from that of H. gramineum on barley in its origin. They describe its formation by the anastomosis of the foliar spots /

spots which are greyish brown in colour. These are stated to originate as chlorotic areas with reddish brown centres, which lie towards the margin of the leaf above the junction of the blade and the sheath. The reddish brown spots become purple brown to grey in 21 days and coalesce to form the stripe. They have recorded the early symptoms on the coleoptiles as small brown spots. The disease is mentioned to exist in two stages, the primary and the secondary stage. In the absence of a severe attack the plant recovers from the primary attack after shooting the fourth leaf which is spotless. The secondary infection is brought about by conidia formed on dead leaves, the victims of the primary attack.

From the above historical survey of the disease we find no agreement between any two statements regarding the causal organism and the disease of oats. Although Johnson and Ritzema Bos diagnose the causal species as H. gramineum yet it is generally accepted that the disease of oats is caused by H. avenae only. Dreschler in his accounts also expresses doubts as to the statement that there is only one species parasitic on oats, but no later account confirms his doubt. Now the question arises whether really there are two separate organisms responsible for the stripe and spot disease of oats respectively or whether these two symptoms are a constant feature of the disease caused by the same organism exhibited under different conditions ? It is the chief aim of /

of this work to answer this question. Other questions of especial importance to a farmer such as the control of the disease are also considered. The mode of attack of the fungus must also be discussed. Ravn suggested the Smut analogy, but so far as H. gramineum is concerned in barley this analogy is rejected by Smith. As for H. avenae, Turner and Millard reject both the theories put forward by the above workers and attempt an explanation of their own, which I beleive is right and in complete agreement with Smith although they claim to differ. A detailed account of their claims and justification to differ is given else-where in this paper and the lines of approach to a complete agreement between these workers is suggested. This is a question of vital importance as on it must be based the general principles of the control of the Helminthosporium disease.

2- Distribution of the Organisms.

These organisms are reported to occur practically every where in the cereal growing areas where oats is the principal crop. It is recorded in all countries, in England by Smith, Turner and Millard, Scotland by O'Brien and Prentice, Germany by Eidam and Rathschlag, Denmark by Ravn, Holland by Ritzema Bos, Austria by Hecke, Italy by Briosi and Cavara, Japan by Yoshino and Ito, India by Butler, America by Dreschler, Johnson and others. It has also been recorded in Canada, Australia and West Africa.

In Scotland it occurs periodically affecting the young crop and causing the loss in 'braird', which in certain cases leads to a total failure of the crops. Examination of the oat fields this year revealed the presence of the fungus in almost all the fields examined in the eastern counties. Both the stripe and the spot forms are seen in the fields. The actual damage to the crop is not so serious in the south east as it is in the north and north west.Some fields in Perthshire and Fife were greatly damaged, the crop being stunted and thin. The actual damage to the grain was not so pronounced in these counties as in the east Lothian where in certain fields the number of the bleached empty heads reached upto 15%. In Perthshire although the grains were formed, they were brownish, shrivelled and light in weight. The disease becomes epidemic near the harvest time and a large /

large percentage of the plants is infected. The table given below is an account of the examination of grain samples obtained from various parts of Scotland.

1*	2*	3*	4*	5*	6*
Black lortana.	65	Black and shrivelled & light in weight.	10	3	Fusarium spores.
Victory	73	Clean.	10	4	
Sandy	82	Clean, few grains light.	10	2	
Abundance	93	Clean	10	1	
Unknown	79	Brownish, shrivelled & light.	10	5	Mycelial knots.
Victory	90	Brownish, and light in weight.	10	6	Fusarium spores.
Victory	87	Few grains brownish.	7	3	
Unknown	92	Grains shrivelled.	10	7	Fusarium spores.
Marvellous	95	Clean.	10	3	
Unknown	89	Clean.	10	4.	

1*. Variety of oats.

2*. Percentage germination.

3*. General condition of the seed.

4*. Number of seeds examined.

5*. Number of spores of Helminthosporium present on the seeds examined by washing the sample in water in a watch glass and counting the number of spores in water.

6*. Spores of other fungi if present.

Note:- Mycelium of Helminthosporium present in the pericarp /

pericarp of all the infected grains from all the seed samples examined. The figures in the column 5 are only approximate, as it is impossible to count all the spores in a volume of water which is comparatively large. And more-over some spores remain sticking to the grains even after washing.

The table on the previous page shows that the fungus is largely distributed all over the country, and in fact I have not been able to find any field or sample of seed which can boast of its freedom from this fungus. The importance of Helminthosporium as a parasite is very great as the yield of the crop may sometimes be reduced upto 80 %. In a country like Scotland where the chief cereal crop is oats this loss in yeild by 20 % is very great and in many seasons counts heavy on the farmers.

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3- Sources of Infection.

Observations show that the Helminthosporium disease of oats occurs in two phases, the primary and the secondary phase. The primary phase results from the infection of the seedling either from the seed or soil. The infected seedling dies in certain cases and forms the source of inoculum for the secondary infection. The actual effect of the fungus invasion will be dealt with under the symptoms of the disease. The secondary infection in a certain field is more or less dependant on the intensity of the primary attack. However, it can become serious even in the absence of the primary outbreak if the adjoining fields are infected. It has been shown in America that the spores of these fungi are carried away long distances by wind and such spores remain viable for a good length of time and are capable of infecting the host under suitable conditions. So the importance of the secondary phase cannot be neglected. Since the production of the inoculum for the secondary infection results from the primary attack, the control of the latter in a certain area will be useful, and the chances of the appearance of the disease in its secondary form are remote if practically complete control of the primary outbreak be obtained.

An examination of the grains as reported on page 14 indicates that the fungus can be carried along with the seed from year /

year to year in the form of spores. Such spores when tested show a very poor percentage of germination and hence these spores on the outside of the seed are not very important as the causal agents of the disease. Even if they were viable in fairly large numbers their importance cannot be very great as it has been proved by infection experiment that spores on the outside of the grain cannot succesfully infect the host in large numbers. However, the mycelium of these fungi is found in the inner layers of the seed coat. This mycelium remains viable for atleast two years and can be obtained in culture for verification. Certain mycelial knots, also sometimes known as pseudo-sclerotia, are observed below the pales covering the seed proper. So we find that the fungus can be present along with the seed in the form of spores, mycelium and mycelial knots. As will be made clear later under the mode of the infection of the organisms, it is not the mere presence of the fungus which is important in producing a diseased plant, but it is its position that counts.

In the infected grains the fungus is present in practically all parts of the seed proper, the extent of invasion varying with the time of the attack. As the infected seed forms the primary basis on which practically all infection rests, it is advisable to study the position of the fungus in the various parts of the seed proper. In order to have a clear conception of the whole position a short account of the /

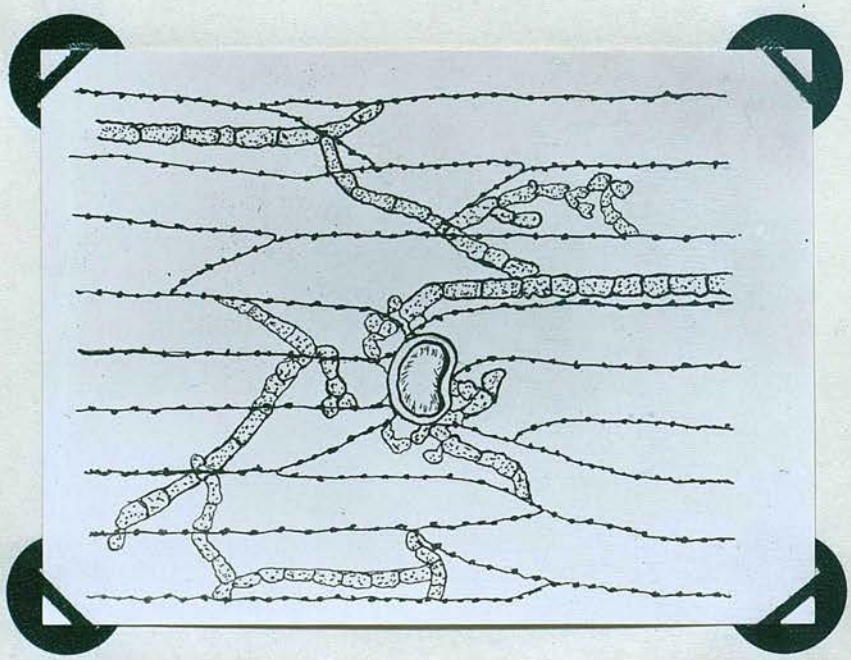

Fig.1. The mycelium of Helminthosporium avenae in the cells of the outermost layer of the pericarp.

the seed structure is given below.

The oat seed may roughly be considered to consist of the pales and the seed proper. The pales cover up the seed proper on almost all sides except the brush end of the seed where a short passage is left. In between the seed proper and these pales a cavity is left which in most cases is the seat of the fungus. Moisture is retained in this cavity by capillary force while the crop is standing and the mycelium does not dry out. This mycelium is harmless after the crop is harvested, as it eventually dries up in the dry places where the seed is stored. The grain proper may further be divided into the embryo and the endosperm, both being surrounded by the pericarp. The pericarp consists of an outer layer of elongated, more or less rectangular cells with beaded walls. Fig. 1 . The long axis of these cells lies parallel to the long axis of the grain. On the outside of this layer are found hairs distributed all over the grain, but they are aggregated more towards the so-called brush end. Below this layer is the cross layer of cells which in mature grains is almost crushed. Then a few layers of similar cells follow, but the cells in all these are crushed and can rarely be distinguished separately. The cells of these layers are parenchymatous in nature and the thickness of these layers, which lie at various angles across each other, varies at different places. All these layers are sometimes collectively known as the pericarp or the seed coat. The testa /

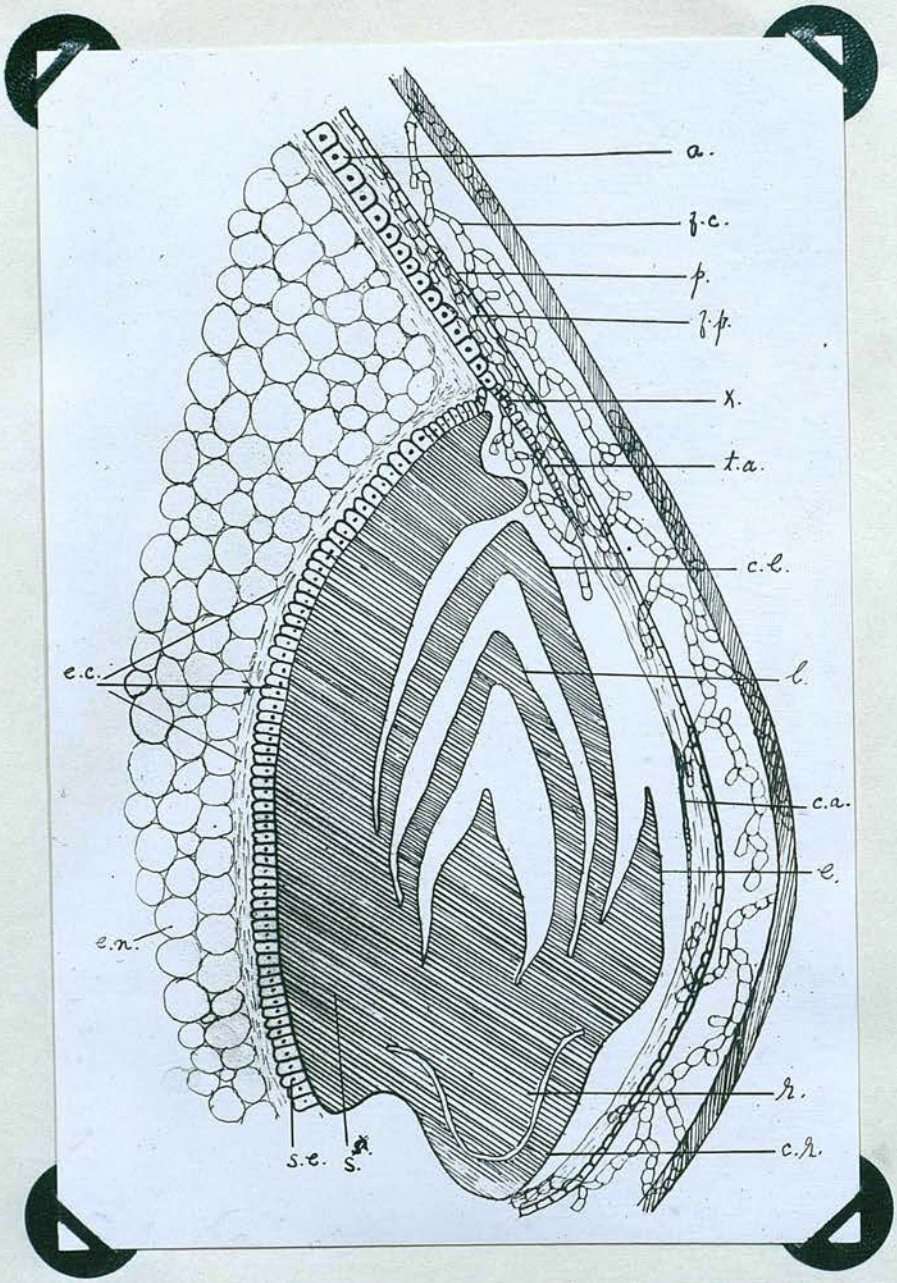

Fig. 2. Section through the embryo of an oat seed.

testa of the seed in mature grains cannot be distinguished from the rest of the crushed layers of the seed coat.Below these layers lies the aleurone layer which consists of thick-walled cells. These cells contain the aleurone grains which in certain places are the only proof of their presence.This layer is continuously thick all round the grain except where it faces the embryo. Here the cells are thinwalled and can easily be penetrated by the fungus. In fact in certain cases their identity cannot be made out on this side except just opposite the scutellum parenchyma. For further description reference may be made to Fig. 2 . which represents a vertical section through the embryonal end of the grain. The cavity between the grain proper and the pales is seen towards the outside and in this are seen numerous hyphae f.c. Then comes the seed coat or the pericarp p with the fungus f.p., followed by the aleurone layer(a) which is normal opposite the endosperm, and thinwalled (t.a.) opposite the scutellum parenchyma, and crushed (c.a)opposite the embryo. The embryo proper comsists of the primaty bud ensheathed by the first leaf (l) and the coleoptile (c.l); and the primary root (r) ensheathed by the coleorhiza (c.r).The embryo is connected to the endosperm by the scutellum which consists of the scutellum parenchyma (s) and the scutellum epithelium (s.e). The endosperm consists of normal parenchymatous cells (e.n) surrounded by the crushed layer of the endosperm (e.c). At the junction of the coleorhiza and the coleoptile is the epiblast (e) which /

which forms a part of the embryo. The portion connecting the shoot and the root is called the crown. The micropyl cannot be distinguished in the mature grains.

With a rough idea of the structure of the seed we can proceed to discuss the position of the mycelium in its various parts. As stated the infected seed may have the fungus on it in the form of spores adhereing to it on the outside, or in the form of mycelium in the seed coat or the pericarp and the pales, or mycelial knots in the cavity mentioned above. Now we will consider them one by one.

Spores:- Considering the conditions under which the spores are found we see that they are always exposed to atmospheric dessication during the storage of the seed. So usually they dry up and rarely remain viable after a few months of storage and are therefore not very important in causing the disease. Even if they remain viable, their importance is not very great as will be shown when we come to consider the mode of the attack of the organisms. So they can be regarded as almost harmless as compared with the mycelium which is the chief agent in causing the disease.

Mycelium:- The most important form in which the fungus perennates along with the seed is the resting mycelium found either in the tissues of the husk (pales) or in the cavity between the husk and the grain proper, or in the pericarp and between the pericarp and the aleurone layer, or in the endosperm. This mycelium remains viable upto a couple of years in the seed coat and the endosperm and is always in a position /

position to attack the young embryo under favourable conditions. In certain cases when the mycelium reaches the embryo the latter is killed and cannot resume its activities and the seeds do not germinate, the result being a poor stand. The mycelium in the pericarp cells and that lying between the aleurone layer and the pericarp is important in producing diseased plants. That in the endosperm usually kills the embryo early and thus no plant (at all) is produced. This mycelium is dark coloured and its hyphae are thick-walled, highly septate, with almost spherical cells in certain places, and the hyphae usually lie parallel to the long axis of the cells of the pericarp. In these cells the hyphae when passing laterally to an adjoining cell row penetrate the walls obliquely (Fig. 1), and may show aggregation before passing through the cell wall. The cell contents of this mycelium are granular. When plated on malt agar it soon resumes activity. This type of mycelium is also found in very old cultures on potato, malt and oat agars. The aleurone cells have not been found to contain any mycelium of this type. This mycelium however shows aggregation near the base of the hairs and in the vicinity of the upper end of the scutellum. In one case a hypha has been traced into a hair, which indicates that it is through the hair that the infection of the seed occurs. But this is merely a supposition as it does not conclusively decide that the hypha travelled from the hair down-wards and not from below into the hair.

The mycelium in the tissues of the husk (pales) has the same importance and is equally in a position to attack the coleoptile and coleorhiza when these have ruptured the pericarp. The dark mycelial knots which are also present on the grain in certain cases are also of the same value as the mycelium lying in the cavity between the husk and the grain proper. They are merely balls of mycelium having hyphae which in structure resemble those in the pericarp cells and are of little or no importance in producing disease. These superfecial hyphae are very brittle and break irregularly into small fragments when pressed under a coverslip. No perithecia have been found on the grains.

The fungus may also be present in the soil and on the straw in as many forms as it is present on the grains. Spores, mycelium and the pseudo-sclerotia have been found on straw in farmyard manure. Further the mycelium develops and spreads freely in the soil in a saprophytic form. Consequently this mycelium is always present in the manured soil when the crop is sown, provided that the manure contains some infected straw. But as already explained the grain itself is almost always infected and in consequence soil infection becomes a matter of little importance.(Under certain conditions this soil mycelium,however,is quite important.) The ploughing activities of the farmer also tend to destroy the soil mycelium by turning up the soil and killing it by drying. The importance of this mycelium in the primary infection is not /

not very great as will be explained later under the mode of infection of the organisms. It is however very important as regards secondary infection. It produces spores on straw and humus which are carried away by wind to long distances and thus help in spreading the disease.

4- Symptoms of the Disease.

The primary study of the symptoms was made from the naturally infected plants and then confirmed by artificial infection from pure single spore cultures obtained from various sources. The symptoms fall into two main groups. The first may be taken as the 'Primary Leaf Spot' formation while the other as the 'Primary Leaf Stripe' formation.There are minor differences within these two groups as will be indicated later, but all the resulting symptoms produced by the various isolants of the organism can safely be classed under one or the other set without any considerable error in the diagnosis.

'Primary Leaf Spot':-

The symptoms of the disease as exhibited by the host depend always on the intensity of the attack and the environmental conditions under which the host plant grows. If the embryo is attacked before resuming activity, no plant results and thus there are no symptoms in the field except thin stand of the crop.Since poor stand may be due to several other causes it does not always mean an attack by Helminthosporium. /

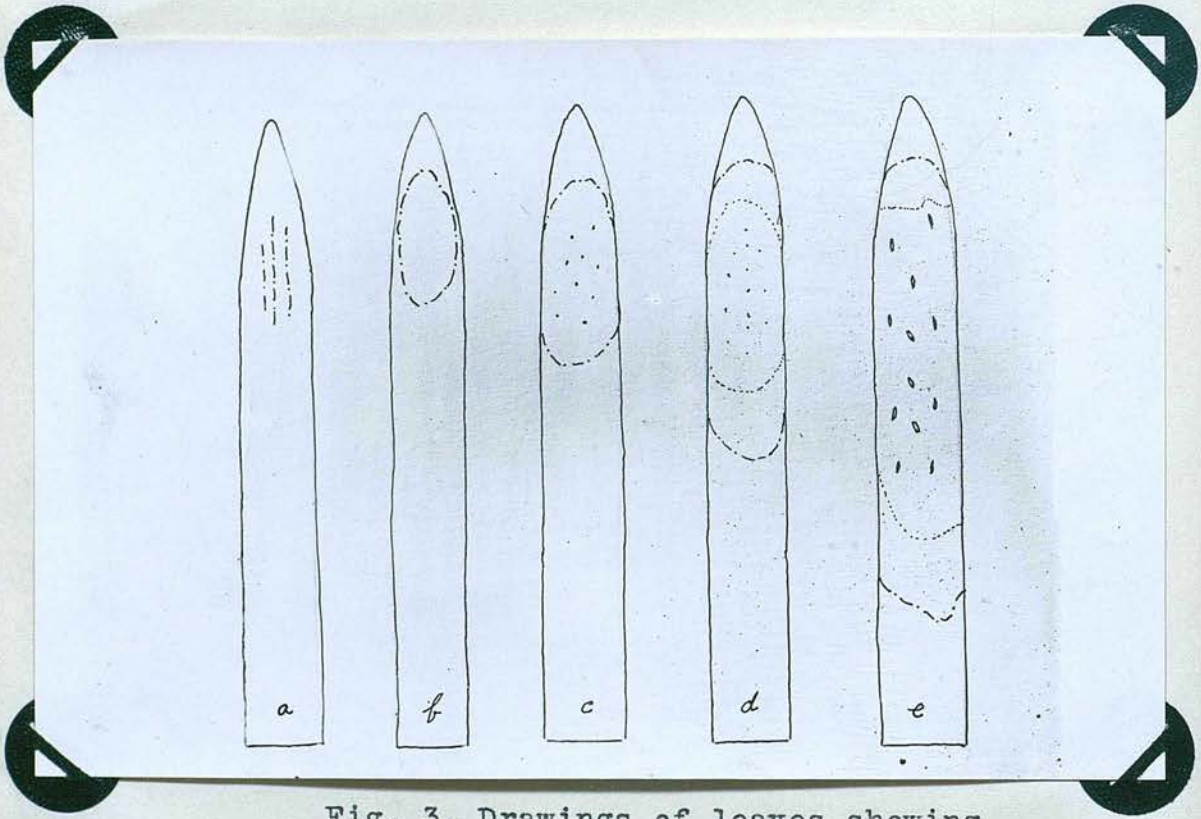

Fig. 3. Drawings of leaves showing primary leaf spots.

The embryo may be attacked severely just after resuming activity. In such cases the plants fail to push up to the soil level and again poor stand results. The third case is when the young plant is attacked at its growing point but owing to sufficient food has managed to push up to the soil level. Such plants die soon after emergence and sometimes this phase of the disease is spoken of as 'Seedling Blight'. In the above three cases one cannot say without microscopic examination whether the plant has been killed by Helminthosporium or any other agency. Definite spots or stripes at the seedling stage are almost absent in the plants which later show typical symptoms. In the case of the 'Primary Leaf Spot' only those plants can show typical symptoms which live upto atleast one month. Plants that are severely infected form leaves that are abnormally twisted and such plants rarely live to produce the third leaf. So while describing the general symptoms of the disease ('Primary Leaf Spot') we will consider those plants only in which spot formation appears. Such plants show no lesions on the coleoptiles except that they are comparatively yellowish. When the plants reach an age of about one month they begin to show the following signs; Small yellow streaks about 5.m.m. long as illustrated in Fig.3.a. appear near the tips of the first leaves. These extend in length and breadth for about three days, during which time they fuse to form a chlorotic area (Fig. 3.b). Next day small tiny pink dots appear in this chlorotic area /

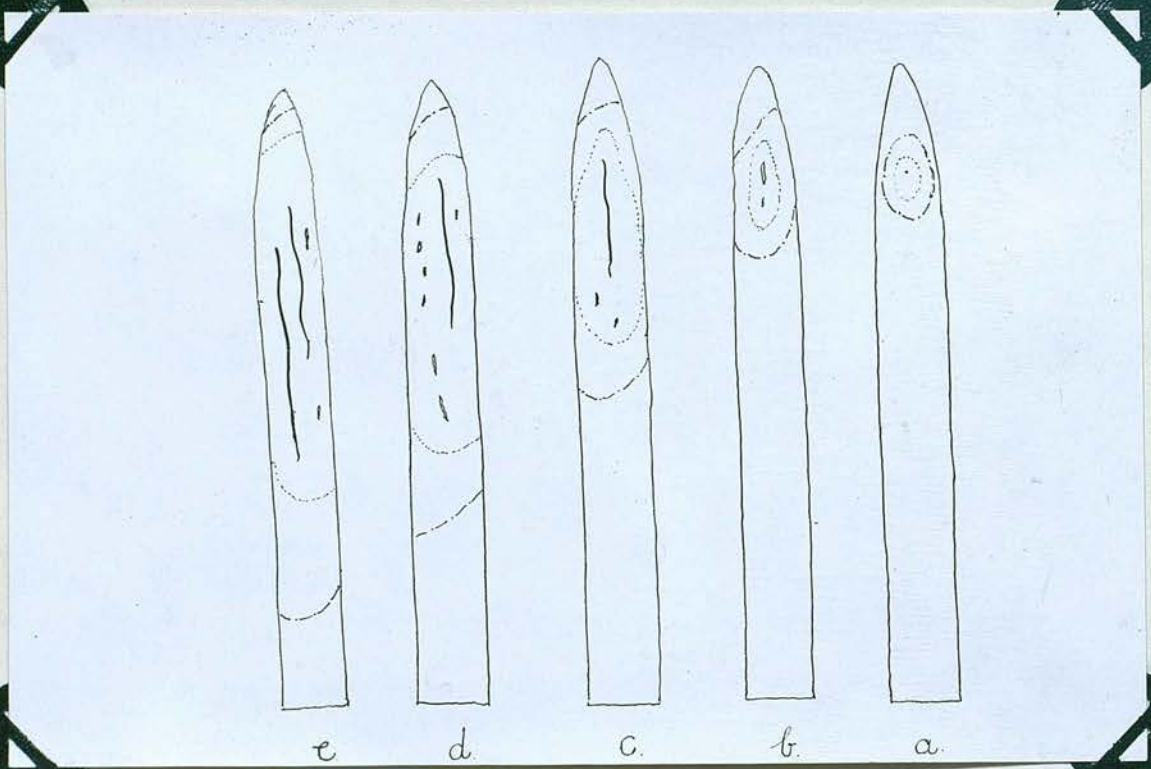

Fig. 4. Leaf drawings. For explanation see text.

area and go on increasing for two days when they attain a deep red shade with a brownish tinge (Fig.3. c.). The area over which they extend is unlimited and is governed by two factors namely, the intensity of the attack and the susceptibility of the host. Within two days these red dots enlarge and become elliptical spots and from them extends out a deep pink halo which slowly melts down to the yellow chlorotic area and this in turn fades into the normal green (Fig. 3.d). The maximum number of red dots counted on a single leaf is 36, but the common number varies between 5 and 10. During the following two or three days these spots increase in size, change shape and colour and become irregularly elliptical with greyish brown margins and bluish grey centres. These spots are still surrounded by a deeper pink area melting down to yellow which by this time extends practically all over the blade (Fig 3. e). Death usually follows soon and the leaf loses its pink pigment but retains the greyish - brown colour of the spots.

If the intensity of the attack is less and the plant strong the fungus spreads following the course as illustrated by Fig. 4. . The illustration 4. a. is almost similar to Fig 3. d., the only difference being the number of spots and the size of the chlorotic area. In the next stage (Fig 4. b) the first spot becomes elliptical as other small pink dots appear and grow and become elongated. In the next stage (Fig. 4. c) two or three of them fuse to form a stripe which is irregularly defined. In this way dots anastomose forming /

forming stripes and in some cases four to five stripes appear on a leaf. By this time the leaf shrivels up and dies as shown in Fig. 4. e. These stripes always originate by the coalescence of the elliptical greyish brown dots or areas and never extend to the sheath part of the leaf, although they always increase downwards till the leaf is dead.

A contrast will be drawn later between the lesions formed by this primary infection and the secondary infection.

'Primary Leaf Stripe':-

In leaf stripe the symptoms are visible from the very begining. Here too we shall consider those plants which shoot out of the soil and shall neglect those which are killed earlier, as the latter are common to both types of the disease and no difference can be made out at such an early stage without testing the organism in pure culture. The coleoptile soon after its emergence shows minute brown stripes (not spots,although some people apparently describe them as narrow and elongated spots) running longitudinally. They are sometimes very prominent and sometimes are almost too faint to be diagnosed at this early stage. At this stage it may be borne in mind that certain varities of oats have a pair of dark bundles in the coleoptile which may be mistaken for stripes. But their opposite position and prominence allows no mistaking by an experienced man. The first leaf which commonly shoots out in such cases at an angle shows a definite pale brownish streak extending onto the sheath and lies on the same /

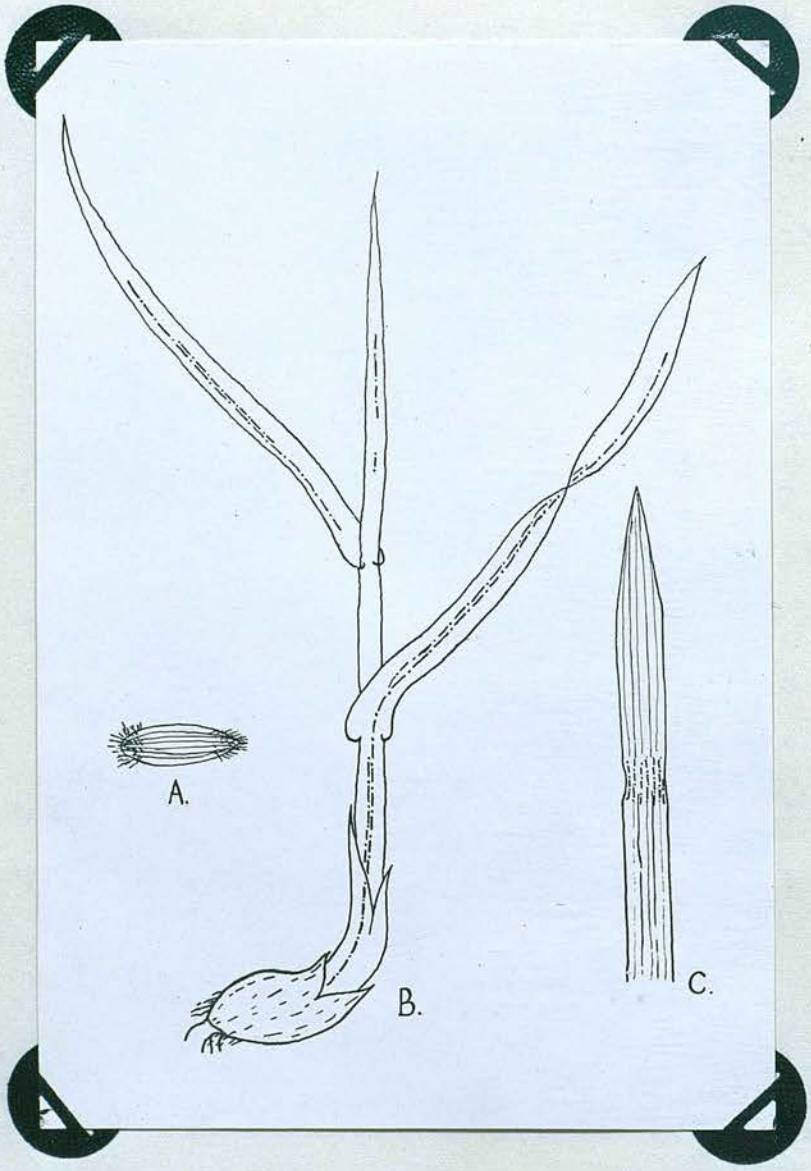

Fig. 5. An oat plant showing typical leaf stripes in illustration B. A, showing a germinating grain and C a leaf with a secondary infection spot.

same side as the stripe on the coleoptile. The stripe is very regular and the common number is one, although three or four may be witnessed in rare cases. Similarly the second and the third leaves show corresponding stripes on the blades, although those on the sheaths only become conspicuous rather late. A typical plant showing these symptoms is illustrated in Fig.5. b.

The stripes do not extend beyond this and the fourth leaf,if it ever shoots out,is invariably free from the disease. Severely infected plants may die at this stage and the stipes which are mature by this time attain a dark grey colour.

'Secondary Infections':-

The organisms responsible for 'leaf spot' disease and the'leaf stripe' disease are capable of producing secondary infections. Such infections are brought about by spores produced on the dead leaves under moist conditions, such leaves being the victims of the primary attack on the plant. Under humid conditions of the atmosphere straight, rarely branched, hyphae come up from the lesions and act as conidiophores, bearing cylindrical, olivaceous, multiseptate spores rounded at the ends with a definitely differentiated basal cell and a well marked hilum. Such spores are blown off by wind and come to rest on the upper leaves and ears of the host. There they germinate giving out several germ-tubes which penetrate the epidermis of the leaves or enter through the stomata. /

The fungus establishes itself in the plant tissue which becomes brownish grey or black. The spot so produced increases in size as the fungus makes its progress in the tissues and has no fixed boundaries. The area surrounding this spot is rarely yellow, commonly retaining the normal green. In early stages in the spread of the fungus the veins of the leaves show a limiting capacity as regards its advancement breadthwise. Thus the spot in early stages is elongated, but can never be confused with a stripe. Later on, however, the barriers are broken through and there is no marked resistance of the veins as regards the penetration of the fungus breadthwise. The number of such spots varies between 5 and 10, and is always controlled by certain factors such as heat, moisture, and the amount of inoculum (spores) available. The disease is most severe when the primary infection is abundant and when consequently there is an abundant formation of conidia. High temperature and moisture also help the fungus since under these conditions the spores germinate freely and there is very little danger of their drying up. If the weather is cool and dry the disease is almost checked even in the presence of an abundant primary infection. The colour of the spots also differs on different varities of oats ranging between reddish brown and brownish grey.

These spots rarely anastomose and usually kill the leaf before they have any chance of union. A leaf showing a single spot which has spread throughout the width of the leaf is /

is illustrated in Fig. 5. c.

The difference between the primary leaf spots and the spots caused by secondary infection may be summarised as follows:-

(i). The spots caused by the primary infection are elliptical and greyish brown with regular boundaries, while those formed by the secondary infection are irregular without any fixed boundaries and have a comparatively lighter colour.

(ii). The primary spots always make their appearance in the chlorotic area and later on are surrounded by a pink coloured area which slowly merges into yellow, while the secondary spots are rarely surrounded by a chlorotic area and the latter when present is only seen after the appearance of the brown spots and never before.

(iii). The primary spots are always found near the tips of the leaves while the secondary spots are scattered all over the leaf surface.

(iv). The primary spots in certain cases anastomose to form an irregular stripe, while the secondary spots never form a stripe.

(v). The primary infection spots do not appear beyond the fourth leaf, while the secondary ones usually begin at this stage.

It may be mentioned here that certain non-parasitic spots are found on the foliage of oats especially in well developed crops. /

These spots are usually lighter in colour and have well marked boundries. In certain fields they appear in large numbers if the weather is rather moist. These spots do not seem to interfere with the activities of the oat plant and the foliage keeps its normal green colour around them. They gradually increase in size but not so rapidly as those caused by Helminthosporium. All attempts to isolate any fungus or bacterium from them have so far yeilded negative results. In certain fields the green-fly has been seen causing damage to the crop and forming exactly similar spots on the foliage as are formed non-parasitically. But this cannot account for the large number of spots found in certain fields in the absence of the green-fly. There certainly seems to be something responsible for these spots, but whether it is the result of some parasite or merely physiological disturbances it is unwise to say at present.

Infection of the ears:-

The ears (panicles) are also infected in the same way as the leaves. If the infection is early enough, there is no grain formed and the fungus spreads down the stalk of the infected spikelet and thus infecting the neighbouring spikelet. Infected spikelets are usually bleached. However, if the infection is rather late, pale brown to chocolate brown spots with irregularly defined margins appear on the glumes. In those cases where the grain proper is infected the seeds are lighter in weight, brownish and shrivelled. Such grains are lost in /

in thrashing, thus greatly reducing the yield. If however the infection takes place near harvest time the endosperm of the grain is not reached and only the pericarp is infected, the aleurone layer acting as a barrier for the further spread of the fungus. Some writers are of opinion that it is the crushed testa that acts as the barrier for the further spread of the fungus in the almost mature grains. From our point of view it is immaterial which layer acts as the barrier, so long as the fungus does not spread into the endosperm. Such infected grains are the carriers of the fungus from one generation to the other, and are rather more dangerous in spreading the disease than those which are badly attacked. The latter produce no plants and thus do not spread the fungus. The infection of the ears is also controlled by temperature and humidity. Wind also plays a considerable roll in the spread of the spores. Infection is most severe if the weather is moist and windy just after the flowering season. At this time the young fertilised ovaries are badly attacked and sometimes all the panicles in a crop become blighted, a stage commonly called the 'White Heads' by the farmers. Sometimes one finds the statment in literature that the actively growing parts of the plant are not infected by the fungus and that the fungus attacks those parts severely that have attained full growth. This statement is very misleading as observations show that the fungus does not spare any parts whether young or old and attacks them with the same vigour.

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5- Isolation of the Organisms.

Not less than five different strains of the fungus have been isolated from various sources, each strain differing from the other in certain points that will be mentioned later under the characters of the organisms. A considerable number of leaves and grains which were attacked by Helminthosporium was incubated either in Petri dishes with moist filter paper at their bottom or in tubes with sterile cotton wool pads at their bases. The dishes and the tubes before use were perfectly sterilised in an autoclave and sterile water was used to supply abundant moisture. The grains and leaves were surface sterilised before incubation by dipping in .1% .2% mercuric chloride for about half an hour and then in a very dilute solution of formalin in 50% alcohol and finally washed thoroughly with sterile water. From about 80 % of the leaves and 60 % of the grains the mycelium of Helminthosporium came out and this was transferred to plates of malt agar. The tubes and plates containing the original material were kept for about ten days in order to examine the spores formed.

In this way in all 63 cultures were obtained throughout the work which could be clearly separated into five sets. Each set is considered to be a distinct strain of the fungus.

For detailed study of the cultural characters single-spore cultures were derived from all the five sets of strains by /

by picking the spores with fine sterile needle and plating them on to a hanging drop of malt agar. Picking was done under the low power of the microscope and in order to make sure of the presence of single spores in each drop the agar drops ,too, were examined before incubation. All the hanging drops were had on the coverslips and these were inverted over glass rings. The coverslips and the glass rings were sterilised before use. The rings were placed in sterile Petri dishes after the spores were transferred to each drop.

The spores of each strain were obtained by inoculating living seedlings of oat under sterile conditions. Surface sterilised grains were sown in wide test tubes with a pad of cotton wool at their bases. The tubes were sterilised before use. When the seedlings were about two inches long they were again surface sterilised by dipping in a solution of mercuric chloride and subsequent washing with sterile water. Such surface sterilised seedlings were put in sterilised pluged tubes and inoculated with a bit of the mycelium from a pure culture of the fungus. In this way about half a dozen tubes were prepared from each strain and kept for about ten days. Sterile water was liberally supplied to all these tubes and within a fortnight spores of Helminthosporium were formed on the seedlings.

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6- Characters of the Organisms.

Aerial mycelium:-

Strain. A. Fluffy white, turning yellow with age and showing traces of pink at places in almost all the cultures. About 1.c.m. high.

Strain. B. Resembles that of A, with the only difference that it is less abundant. About 5.m.m. high.

Strain. C. Greyish white and producing the pink colour in the hyphae much earlier than any other strain. Less abundant and about 3.m.m. high.

Strain. D. White and arising in club-shaped tufts, remaining white even at old age. No pink colour produced. About 1 5.cm. high.

Strain.E. White and fluffy and not arising in tufts. About 1.c.m. high. No pink colour produced.

Submerged mycelium:-

Strain. A. Dark greyish when young, becoming black with age. After a few days a pink colour always precedes the dark colour formation in the hyphae and forms a sort of ring about the black area.

Strain. B. Dark green, turning black with a greenish tinge when old. Four definite zones of colour are observed begining from the centre in the order stated, black, reddish brown, green and white.

Strain. C. Dark green as in B, but no zonation is observed. After a few days the red colour precedes the formation of the /

the black colour and the tips are always white. The central dark area is much larger as compared with that of B.

Strain. D. Dark in the middle surrounded by a lighter coloured area having a brownish tinge, the latter being six times as large as the central dark area. No red colour produced in culture.

Strain. E. Almost the whole mycelium is dark, the tips alone being hyaline. The fungus grows more rapidly than any other strain. No red colour in culture.

Spore formation:-

Strain. A. No spores produced on ordinary culture media. Spores produced on straw and malt agar under highly humid conditions.

Strain. B. The same as A.

Strain. C. No spores on any culture medium under any conditions. Spores produced only on artificially inoculated plants.

Strain. D. No spores in culture. Spores produced only on inoculated plants.

Strain. E. The same as D.

Note:- In strains A,B the spore formation on culture media is vastly increased if the cultures are kept in sunlight and provided with abundant moisture. The tendency to form spores decreases as the organisms are kept in culture for a longer time.

Perithecium formation:-

Strains A,B, and C. No sclerotia or perithecia /

perithecia are observed in culture or on natural material in these strains. Hyphal aggregations are of qommon occurance, but these are by no means definite in shape and cannot be mistaken for perithecia.

Strains. D,E. Definite immature perithecia are observed in culture on malt agar, oat agar and straw. Although no ascospores have been observed these bodies are quite distinct from sclerotia.

Types of lesions (Primary) formed on the host:-

Strain. A. This forms reddish brown, elliptical spots at the tips of the leaves surrounded by a reddish pink area which slowly merges into a chlorotic area and f finally into the normal green. The extreme tips of the leaves are always free from the disease.

Strain. B. The lesions are similar to the above but they anastomose to form an irregular stripe which acquires a greyish brown colour when old.

Strain. C. This forms brownish red, elliptical, rather elongated spots which turn grey with a brown tinge at the margins and a bluish tinge in the centre. These are also seen first near the tips and fuse to form an irregular stripe.

Strain. D. This forms long,narrow,greyish black, regular stripes resembling those formed by H.gramineum on barley, the only difference being that the leaves do not split. The stripes develop succesively on the coleoptile, first, second /

second and the third leaves.

Strain. E. This forms short, broad, brownish grey stripes which do not extend over the whole leaf, but are commonly confined to the lower part. They are very regular on the coleoptile and first, second and third leaves.

Source of isolation:-

Strain. A. Grain.

Strains. B,C,D, and E. Leaves.

The description of the aerial mycelium as well as the submerged mycelium is given from cultures on malt agar. These cultures were incubated at 25°C. and were a fortnight old when they were described. The structure of the hyphae is similar in all cases.

From the above account at a glance we can divide the five strains into two chief groups fundamentally different from each other in the sense that the strains A,B,C form spots on the leaves and do not form any perithecia in culture under the conditions tested, while strains D and E form stripes on the leaves and form perithecia both in culture and on naturally infected material. Minor differences serve to distinguish individual strains. From the characters given above I feel justified in dividing the species into two sub-species namely, H. avenae sativae and H.avenae graminae.

Before proceeding on to the general characters of the organisms such as morphology, it will be worthwhile to record here a few more facts which have not been given before in the systematic /

systematic comparision of the five strains.

It has been noticed that when freshly cut leaves naturally or artificially infected with the stripe strains are incubated under similar conditions of temperature and moisture, there is a remarkable difference in the mycelial growth obtained. From the leaf infected with strain D the white mycelium arises in well organised tufts which are club-shaped and about 1·5 c.m. high; the mycelium in this case grows out from the tissues surrounding the stripe and not from the stripe proper. In the case of leaves infected with strain E, the mycelium comes off irregularly from all parts of the leaf and does not stand in tufts, but is rather woolly and thin. There is no remarkable difference in the mode of emergence of the mycelium in the case of strains A, B and C.

Another character shared only by the strains of leaf spot group is the production of the red pigment in the aerial as well as submerged hyphae. This colour is invariably produced on all media, except plant tissues which are inoculated after they are dead. On living tissues such as leaves the mycelium turns red within two days after its emergence under moist conditions. Within a week conidia of the Helminthosporium type are developed and the colour of the mycelium becomes brownish red.

The ideal conditions for the production of the red colour are the abundance of moisture, light, and low temperature. The /

The tendency towards red pigment development increases with the time during which the organism is kept in culture. Very old cultures do not become black for a long time but only produce a reddish colour. Certain cultures may remain perfectly red for two or three generations, after which they again develop the black colour.

Two interesting facts have been observed in the behaviour of strains A and C.

When strain A is exposed to the vapour of alcohol the whole culture turns red within 24 hours. Alcohol vapour can be supplied to a culture in a Petri dish by cutting out a bit of the medium and putting one or two drops of alcohol in the pit thus formed. The lid is then replaced, and the alcohol vapourises into the air of the Petri dish. The cultures also develop more red colour on media with high carbohydrate content. The above two facts suggest that in nature the fungal hyphae by their reaction on the host tissue produce certain enzymes, hydrolytic in nature, which hydrolise the sugar present in the plant cells to form traces of alcohol. This alcohol then induces the production of the red colour in the hyphae. In support of this view it may be stated that the extreme tips of the hyphae are not coloured under any conditions and in nature the pink coloured area sorrounding the greyish brown spots is always surrounded by the chlorotic area. As the hyphae grow they kill the plant tissues producing alcohol which induces the red colour formation. The alcohol /

alcohol only seems to have a stimulating effect. If the alcohol is administered in comparatively large quantities the growth of the fungus is retarded and no colour develops. What bearing this pigment production has on the life-history of the fungus is not clear. However if we go on taking subcultures from the red portion, the resultant cultures produce no spores under any conditions.

Strain C seems to behave in quite a different way. The red portion which sometimes appeared as a sector when grown on maltose salts agar in the fifth generation and kept in light for three days after a week's incubation produced spores resembling those of Dicoccum asperum. The only difference between these spores and those of Dicoccum is that the Dicoccum spores are hyaline while these are dark coloured. These spores are bicellular, dark, with unequal cells and verucose walls and measuring 10-15 X 25-30 μ. The dimensions are in complete agreement with those of Dicoccum asperum. Owing to lack of time further work with this saltant or mutant had to be given up.

The five strains show no differences in the general morphological characters and in consequence one description will suffice for all of them. The aerial mycelium consists of thin-walled, septate, usually hyaline or pink hyphae, 3-5μ thick, with no constriction at the septa, but showing abundant vacuolation. The mycelium is usually fluffy, its amount increasing with the abundance of moisture, and under these conditions in /

in certain cases it may tend to collect in clusters. Its amount also increases when grown on media with high carbohydrate content, the inoculum being the aerial mycelium.

The submerged mycelium consists of thick-walled, highly septate hyphae which are commonly dark and 6-7 μ thick. In young cultures there is no constriction at the septa, but in older cultures constrictions develop and the cells become almost spherical. The colour of the mycelium varies slightly with the medium on which the fungus is grown. The cells are more elongated when grown on malt agar and they are almost spherical when grown on potato agar. This mycelium aggregates at certain places both on artificial media and on plant tissues. The mycelium in the host cells resembles the submerged mycelium in culture when sufficiently old.

The fact that this species of Helminthosporium does not form spores normally in culture led me to try various media in order to get spores of the fungus. The following media were tried:-

(1). Malt agar. (2). Oat meal agar.
(3). Potato agar. (4). Potato-dextrose agar.
(5). Beyrinch's agar. (6). Rice meal agar.
(7). Wheat meal agar. (8). Maize meal agar.
(9). Crushed oat grains. (10). Oat straw.
(11). Fresh oat leaves. (12). Lemco agar.
(13). Cane-sugar salt agar. (14). Levulose salts agar.
(15). Cane-sugar levulose salts agar. /

(16). Lactose salts agar. (17). Dextrose salts agar.
(18). Lactose-dextrose salts agar.
(19). Fructose salts agar. (20). Glucose salts agar.
(21). Maltose salts agar. (22). Peptone salts agar.

All media were sterilised at 20 pounds pressure in the autoclave for about half an hour except the green oat leaves which were surface sterilised by dipping in a ·2 % solution of mercuric chloride for half an hour and subsequent washing with sterile water. In all the synthetic media except Beyrinche's agar where the original formula was followed, the following concentration of the salts was used;

Potassium nitrate.	2 gr.
Sodium chloride.	·5 gr.
Magnesium sulphate.	·5 gr.
Potassium-hydrogen phosphate.	1 gr.
Water (distilled).	1000.C.Cs.
The respective sugar.	10 gr.

The cultures were kept at various temperatures ranging between 5 and 30°C., both in light and darkness. Under these conditions no spores were formed on any medium except straw on which a few spores developed.

The spores of this fungus are sometimes produced in culture on Oat agar and malt agar if the cultures are supplied with abundant moisture and kept in light. Freshly isolated fungi form more spores than old cultures. However, the spores of the strains belonging to the'Leaf Spot'set can easily be obtained by placing bits of culture (culture on malt or oat agar) on /

on moist filter papers in sterile Petri dishes, and placing the Petri dishes in direct sunlight. Spores are produced within ten days and are perfectly normal and capable of infecting oats. The method recommended by Turner and Millard in which the fungus was grown on oat seedlings under sterile conditions was used with success with minor changes as suited to the occasion.

The spores obtained by either of these methods or from naturally infected material are cylindrical, multiseptate, olivaceous, 30-120 μ long and 14-18 μ broad and rounded at the ends. The basal cell is always sub-hyaline, hemispherical with a well marked 'hilum', this being the place of attachment with the conidiophore which is represented by a dark spot. In certain cases the terminal cell is also differentiated from the rest, but generally this cell shows no difference. In those cases where it is distinguished it is lighter in colour and its contents are more granular.

The spores attain their full length before septation and the cell contents are more or less granular and the walls are normally thick. The first cell to be cut off is the basal cell, which remains lighter in colour, while the rest of the cells which are cut off almost simultaneously, attain a dark olivaceous colour. The septa are straight and parallel to each other in most cases, but they may be frequently irregular. The septa are of the same thickness as the outer /

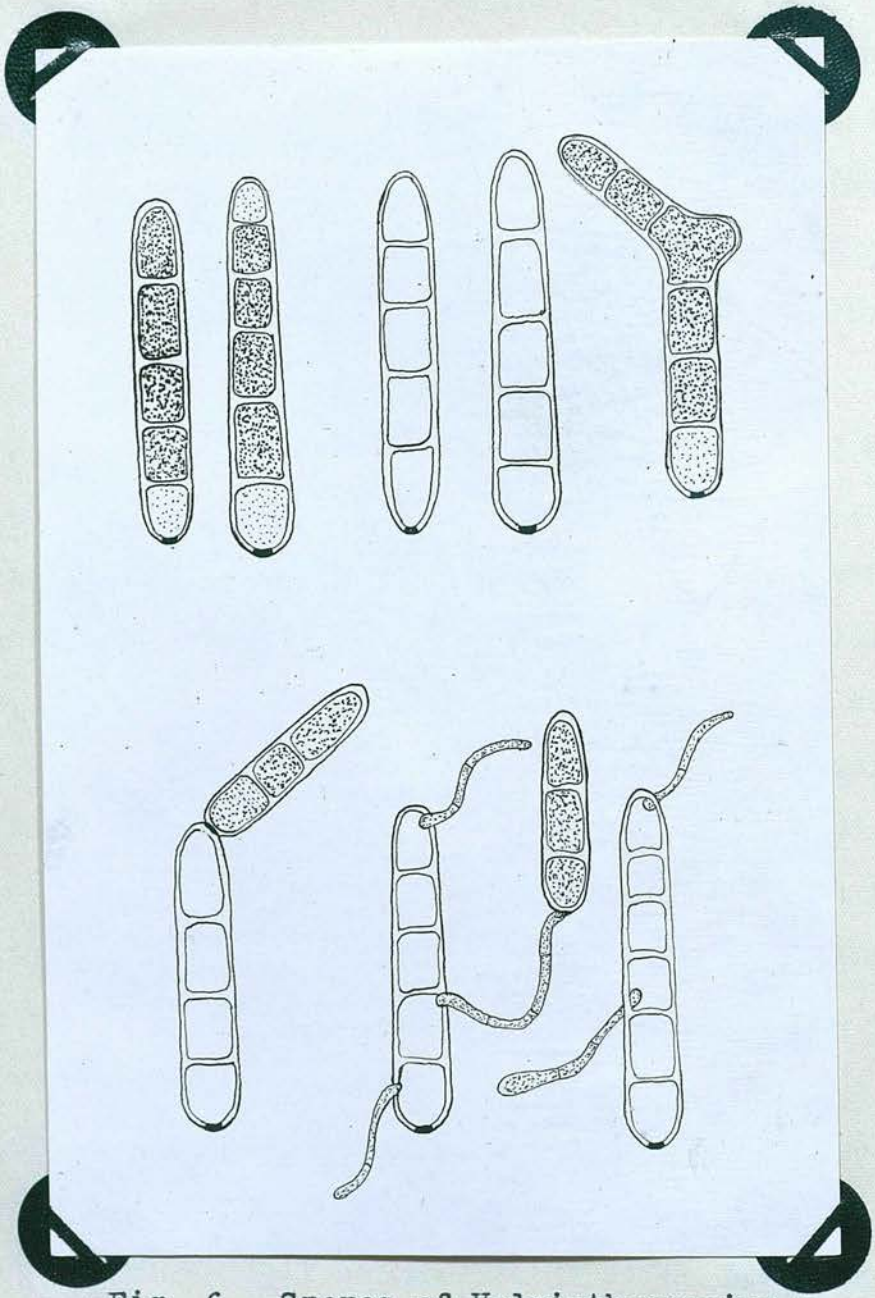

Fig. 6. Spores of Helminthosporium avenae.

outer spore wall and appear elliptical when seen obliquely, thus confirming the cylindrical nature of the spores. The maximum number of septa recorded is seven from naturally infected material and six from artificially obtained spores. The actual shape of a spore varies greatly. Some are cylindrical with almost parallel walls while others taper towards the apex; some are broader in the middle and curved and still others are branched.

The following table shows the measurement of 145 spores with their number of septa and percentage:-

No. of septa.	Length	Breadth	Number of spores.	Percentage (approx.)
6	102-125 μ	16-17 μ	23	15·9.
5	90-110 μ	16-18 μ	14	9·65.
4	67-99 μ	15·5-17 μ	49	34·0.
3	60-90 μ	14-16·7 μ	38	26·2.
2	25-55 μ	14-16 μ	16	11·0.
1	20-50 μ	14-16 μ	5	3·4.

When placed in water the spores germinate within 16 hours. Thin, hyaline germ tubes are given off from almost any cell, usually 4 to 5 arising from a single spore. Each cell is capable of germination, even when separated from its neighbour but itself left uninjured. Commonly the germ tubes become septate a little later, divide into various branches, and develop into a mycelium. In certain cases the spores produce secondary spores, which resemble the parent. Usually they are formed from the apical cell but they can also be produced /

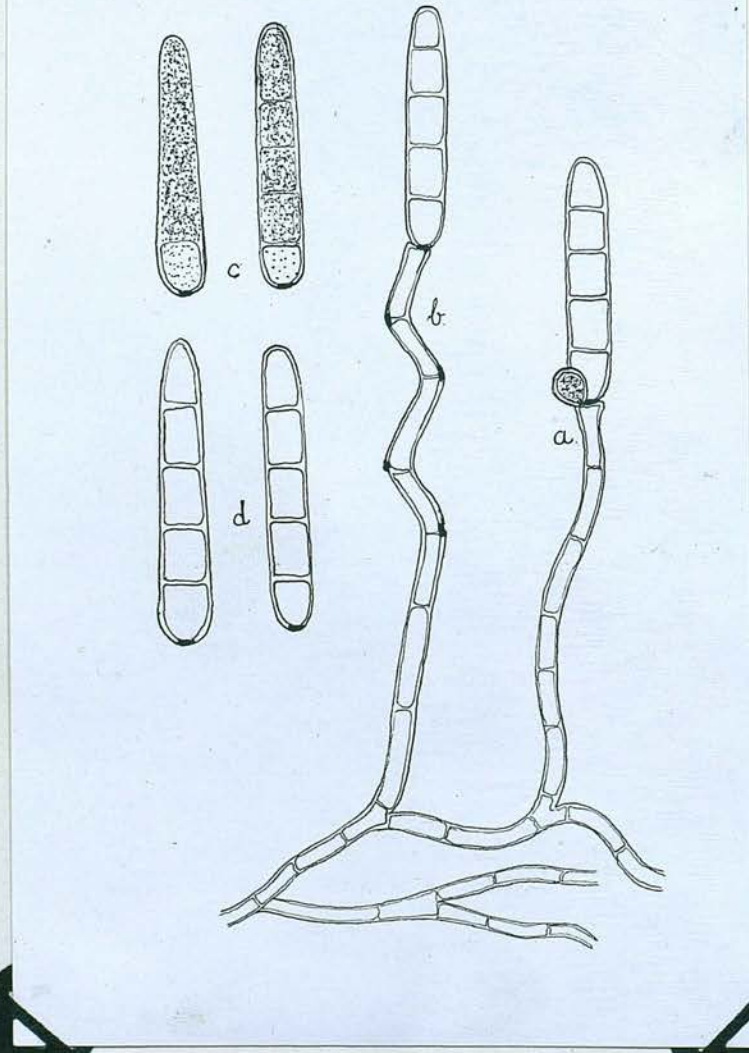

Fig. 7. Conidiophores and spores of Helminthosporium avenae.

produced from intermediate cells in rare cases. The germ-tubes do not elongate in such cases and the spores develop at their ends. Tri-radiate spores which are observed in nature are in certain cases formed in this way.

The conidiophores which bear the spores terminally are olivaceous, thick-walled, multiseptate, with the basal cell sometimes flattened. When the first spore is mature the conidiophore develops a lateral cell which after elongation forms an other spore at its top. In this way the conidiophore becomes kneed at various places where the spores were borne. Branched conidiophores are not very common.The usual length of the conidiophore upto the place where the first spore was attached is 120-220 μ, or 200-450 μ after its full elongation. The thickness is always less than the spore and varies between 12 and 15 μ.From the diseased leaves the conidiophores usually emerge singly, but it is not uncommon to find them arising in pairs or even in clusters. Usually they come out of the stomata or places where the two epidermal cells join end to end.

Although certain differences in size of spores and conidiophores have been observed existing between the two sub-species it is hardly advisable to draw any line between such measurments, the differences being so minute that they have no significance. The conidiophores in the 'Primary leaf spot' group have a greater tendency to branch.

The perfect stage of Helminthosporium avenae has been described in /

in Japan by Ito and in Germany by Rathschlag. The descriptions given by both of them do not agree so far as the measurements and the formation of perithecia are concerned. Ito has been able to get mature perithecia in Japan on straw, while Rathschlag obtained mature perithecia in culture on oat agar. The culture which yeilded mature perthecia was kept at -11° C. for a week. So far, I have not been able to get mature perithecia from the British strains.

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76 Pathogenicity & Infection Experiments.

A preliminary experiment for testing the pathogenicity of the strains A and C was carried out using sandy oats as the host. In all 20 pots (6"X6") of unsterilised soil were taken and sown with surface sterilised seed as follows; 6 pots with the mycelium in the soil at various levels, and four with the mycelium on the seed, two with spores on the seed and eight pots left uninfected. Out of these four served as controls while the other four were inoculated with spores and mycelium respectively when the seedlings were about 1.c.m high. These pots after inoculation were kept under bell jars for about four days and then again placed out along with the rest.

Satisfactory germination was obtained in all cases except where the mycelium was either in the soil or on the seed. In these pots a loss of about 20 % was obtained. In the plants inoculated at the seedling stage infection occured upto 90 %, but the fungus did not spread further and the plants remained almost healthy. From spores on seed infection was absent.

After about a month some plants in the pots inoculated with the mycelium began to show the symptoms as described before and repeated all the stages mentioned. But owing to the partial resistance of the sandy oats the infection was not severe, only a few plants being infected in each pot. /

The preceding experiment being rather rough, the following experiment was carried out under more controlled conditions with all the five strains isolated, using Victory oats as the host. In all 15 pots (4"X4") were taken and filled with sandy loam soil containing fragments of leaves. These were sterilised for one hour at 20 pounds pressure in an autoclave. The pots were thoroughly watered before putting them in the autoclave in order to avoid drying up of the soil. A picked sample of Victory oats showing 99-100 % germination was then taken and surface sterilised by soaking for four hours in luke-warm water and then dipping in ·1 % mercuric chloride for half an hour. From mercuric chloride it was transferred to a very weak solution of formalin in 50 % alcohol for a few minutes and finally washed thoroughly with warm tap water. This seed was again tested later for germination and no loss could be observed. Ten such seeds were sown in each pot. Some of these seeds were dehusked and three controls were sown with normal husked seed and two with the dehusked seed. The remaining ten pots were sown similarly with husked and dehusked seed respectively, and two pots, one having husked, and the other dehusked seed, were inoculated with each strain. The inoculum in each case was placed along with the seed in the soil and consisted of the mycelium grown on oat agar. The pots were placed under bell jars in the green-house and watered with sterile water as required.

All the dehusked seeds germinated and germination commenced two days /

days earlier than in the case of the ordinary seed.Typical stripes appeared on the coleoptiles in the pots inoculated with strains D and E. No marked spots or stripes could be seen in the pots inoculated with the strains A,B and C. All the coleoptiles of the controls were perfectly healthy.

Two days later the husked seeds also germinated, showing stripes on coleoptiles on nine plants out of twenty in the pots inoculated with the strains D and E. Out of these nine plants showing typical stripes, five belonged to the pot inoculated with the strain D, and four to the pot inoculated with E.The rest of the coleoptiles were merely brownish.

The plants inoculated with the strains A,B and C showed merely yellowish coleoptiles but no lesions.

Only two of the husked seeds failed to germinate in all and one of them belonged to the pot inoculated with strain B, while the other belonged to the control.

Within a week five plants died out of the pots inoculated with strains D and E and sown with dehusked seed. Out of these three belonged to E while two to D. All the rest of the plants in these pots showed stripes on their leaves.

No plant died out of the pots inoculated with D and E in which the husked seed was sown. Only seven plamts showed stripes on the leaves although nine coleoptiles had previously shown stripes. The rest of the plants were uninfected. The /

The controls were all healthy and about 1½ times as high as the inoculated plants.

After a month the plants inoculated with the strains A,B and C also showed the symptoms as described on page 23 to 26. Some plants out of these died at various stages. The following table gives the analysis of the diseased and un-diseased plants after six weeks :-

1*	2*	3*	4*	5*	6*	7*
A	Husked	2	5	1	2	30%.
A	Dehusked	0	4	2	4	60%.
B	Husked	3	3	1	2	30%.@
B	Dehusked	1	2	1	6	70%.
C	Husked	1	3	0	6	60%.
C	Dehusked	0	3	3	4	70%.
D	Husked	2	4	1	3	40%.
D	Dehusked	0	2	2	6	80%.
E	Husked	1	3	1	5	60%.
E	Dehusked	0	2	3	5	80%.

@. One seed did not germinate.

1*. Strain. 2*. Seed.

3*. Number of plants typically healthy.

4*. Number of plants showing signs of infection on the coleoptiles, but otherwise healthy.

5*. Number of plants dead.

6*. Number of plants showing typical symptoms.

7*. Percentage infection, calculated from columns 5 and 6. /

The controls all remained healthy. The typical symptoms of the disease were witnessed as stated under the symptoms of the disease. Owing to the small size of the pots the plants could not be carried upto the earing stage. In all cases the plants were cut and several portions from each set of plants were incubated after external disinfection and the fungus was re-isolated in all the five strains from the ~~xi~~ inoculated plants, while the controls gave no fungus except in one case where Fusarium sp. was isolated.

The infection of the aerial parts was carried out with all the strains using spores and mycelium as the inoculum. Leaves could be infected at all stages by the spores as well as the mycelium, the infection being cent per cent in cases where the mycelium or spores were applied under humid conditions and the plants kept under bell jars. In the open, the infection was more severe where the inoculum was applied on to the ruptured epidermis and reached upto 90 %. In cases where the epidermis was sound the infection only occured in those weeks when the atmosphere was wet, otherwise the inoculum dried out.

The ears (panicles) were infected at various times after shooting out and infection could be obtained at all stages if the conditions were kept favourable for the fungus. Ears infected just after shooting did not produce any grain and the chaff became bleached after about a week. Ears infected after pollination developed the grains, but such grains were /

were shrivelled and brownish. The chaffs were markedly spotted in all such cases but were not bleached. After killing the infected spikelets the fungus travelled on to the neighbouring parts and caused infection. The inoculum in the case of the ear infections was always the spores obtained by various methods already described.

No difference could be made out between the lesions formed by various strains in the case of these secondary infections. Strain D was the severest of all and in these experiments yeilded a very high percentage of infection.

From the above experiment some conclusions can be drawn as regards the mode of the attack of the fungus. For instance, we see that the infection in all cases is more severe where the husk of the grain is removed and the inoculum is mycelium. In cases where spores were applied on to the seed no infection could be obtained. This points out to the fact that the spores in nature have a very little chance of infecting where the conditions are rather unfavourable for the fungus. Then moisture is necessary for secondary infection. The actual mode of infection is discussed under a separate heading in the following few pages.

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8- Mode of Infection & Control Measures.

A clear idea of the various stages and possibilities of infection may be obtained by following the course of the disease throughout the life of the host plant. It may be noted here that the fungus reaches the ear only as a result of the secondary infection. Plants infected primarily can only produce ears if they grow out of the disease at the four leaf stage, otherwise they are killed there and then producing no ear at all. The internal structure of the grain infected by Helminthosporium shows that there are several stages during which the developing grain is infected and damage is correspondingly less or more depending on the time and stage at which the infection occurs.

In the first stage the ovary may be infected before fertilisation, in which case no grain ever results and thus this stage need not be considered. The second stage is that in which the fertilised ovary is passing through its various stages of development to the formation of a mature grain. At the stage when all the parts of the grain are formed but are very young, the walls of the cells of the scutellum and of the aleurone layer are not so thick and hard and can easily be penetrated by the young hyphae. In this way the fungus reaches both the endosperm and the embryo and the grains thus formed are light, shrivelled and incapable of germination. In other cases the endosperm may be the only victim;such /

such grains, when sown, also fail to germinate owing to the early spread of the fungus to the young embryo. However, it has been observed that when the endosperm is infected the embryo is also similarly infected and in many cases is dead.

The next stage is when the grain is fully developed but not yet ripe. The fungus attacks the pericarp (See Fig. 2 (page 19) p.) and penetrates its cells easily, but the cells of the crushed testa or the aleurone layer (a) act as effective barriers and, under ordinary conditions, the fungus has no chance of spreading further and reaching the embryo. However, if the conditions are more favourable, it can penetrate the aleurone layer at the place where it just abuts on the scutellum (x). At this place the cells of the aleurone layer are small and comparatively thin-walled, and generally there is a small crevice left where the aleurone layer and the scutellum unite. Mycelium growing down through the endosperm from the upper end of the grain may bridge over the scutellum at this place and so infect the embryo.

But usually the fungus just penetrates the pericarp and grows in it and rests there as a thick-walled mycelium already described. It may be noted here that the fungus can reach the embryo through the pericarp and if the infection takes place about a week before harvest time and the weather be wet and warm the embryo is certainly infected and killed. But in the majority of cases only the seed coat is infected and when such infected seed is sown this mycelium in the pericarp regains activity and grows out into the /

the cavity formed by the husk and the grain proper. In this place it is always in a position to attack the young shoot enclosed in the coleoptile. The coleoptile has to make its way through this cavity and is bound to be infected by the mycelium which is already stuffing the cavity by this time. If the attack is very severe the coleoptile fails to emerge from the husk and is killed. However, commonly the coleoptile emerges, fully infected owing to the thinness and weakness of the cell walls. From here the fungus penetrates inwards infecting the young folded first leaf. Observations show that after the coleoptile has emerged and reached a length of about 2.cms. the seedling can no longer become infected as a whole. The coleoptile at this stage may become infected, but when this is the case the mycelium does not spread within the tissues and the infection does not become general.The fact has been ascertained by infection experiments by inoculating the coleoptile from healthy seeds with mycelium when about 2.c.ms. long. When such infected seedlings are kept under moist conditions the mycelium penetrates the cells and form a lesion, but does not spread sufficiently to injure the developing seedling when the later is transferred to normal conditions of the atmosphere. It may be stated here that it is quite possible to infect seedlings at any stage and obtain the infection in such a severe state that the seedling is killed. But this can only be acheived if the seedling is grown under highly abnormal conditions, a state of affairs not found in nature. /

It may be noted here that it is not possible to have a healthy plant from the seed where the endosperm and the embryo are infected.

So if the young plant be protected at earlier stages when the infection is more severe, it is possible to have a healthy plant, even if the seed be slightly infected. In order to attain this success the following experiment was carried out; Twelve pots (6"X6½") were taken and sterilised after filling with the garden soil. A diseased sample of Victory oats naturally infected by the strain D was taken and tested for germination in Petri dishes. The percentage germination thus obtained by various trials was 86 %, and the number of the diseased seeds was about 40 %. Such husked seeds were taken and 50 seeds sown in two pots; 50 husked seeds treated with Ceresan were sown in another pair of pots. There were 25 seeds sown in each pot. In the third pair the seeds were dehusked and sown untreated and in the fourth dehusked seeds treated with Ceresan. In all these pots the seeds were sown an inch deep and laid flat. The remaining four pots were sown with dehusked seed standing upright with its embryonal end upwards. In one pair they were an inch deep and treated with Ceresan and in the other pair they were only half an inch deep and untreated; in the later case the embryos were almost at the soil level. All pots were watered with sterile water and placed in the greenhouse. The dehusked seeds germinated earlier than the /

the husked seeds. The results of the counts are tabulated below; the plants with first leaf infected being taken as infected.

Table showing the results of the experiment.

	1" deep husked, untreated flat		1" deep husked, treated flat		1" deep dehusked, untreated flat		1" deep dehusked, treated flat		1"deep dehusked, treated upright		1" deep dehusked untreated upright.	
	1	2	1	2	1	2	1	2	1	2	1	2
1*	16	18	18	19	17	20	20	22	21	22	20	22
2*	7	6	3	4	4	5	1	1	0	1	2	2
3*	68 %		74 %		74 %		84 %		86 %		84 %	
4*	38 %		19 %		24 %		5 %		2 %		10 %	

1*. Number of seedlings after ten days.

2*. Number of diseased plants.

3*. Approximate percentage germination.

4*. Approximate percentage infection.

The germination capacity of the sample as determind in the laboratory in Petri dishes was 86 %. From the above experiment the actual germination capacity in the field is revealed to be only 68 %. By just removing the husk we have been able to increase the germination by 16%, and then treating it with Ceresan we have a further increase of 10%. Then a further increase of 2% is obtained by changing the position of the seed, which is reather more remarkable in the untreated lot where there is a difference of 10%. Similarly we have a decrease in the percentage infection by only removing /

removing the husk. Then a further decrease is obtained by treating the seed with Ceresan. If the seed is dehusked and also treated with Ceresan the infection is brought down to only 5%. Then by changing the position of the seed a further decrease can be obtained.

By bearing in mind the results of the above experiment we can have some appropriate means by which we can check the disease on a large scale in the field. The principles of the control are to be based on these results and the results of the infection experiments.

Before attempting an explanation of the results recorded in the fore-going table, its main features may be summarised here again to have a clear idea of what we are dealing with. There are two main features of the table represented in the column 3 by percentage germination and column 4 by percentage infection. Here in the horizontal column 3 we find that the percentage germination is increased to the same extent either by removing the husk or treating the seed with Ceresan. Then further increase in percentage germination is obtained by either sowing the untreated and dehusked seed upright, with its embryonal end upwards, or sowing the dehusked and treated seed flat.

From experiments it is learnt that dehusked seeds or seeds treated with Ceresan, germinate about 48 hours earlier than the ordinary husked seeds when sown in soil under similar conditions. The coleoptile in the case of the husked seed /

seed emerges at the 'brush end' of the grain and has to make its way through the cavity between the husk and seed proper. During the germination of the diseased seed this passage is stuffed sooner or later by the fungus mycelium which grows into it from the pericarp or tissues of the husk. By removing the husk we avoid the infested passage for the coleoptile and so give it a comparatively safe position for further development. By treating the seed with Ceresan we stimulate the growth of the host and also poison the fungus.

The results given under perecentage infection are not similar. This is what would be expected. In the treated seed we are poisoning the fungus and thus clearing the ground for the young host to develop without any interferance. But in the case of the dehusked and untreated seeds we are merely giving a start to the host and are not keeping the parasite down. Since both of them are free to grow the fungus may infect the weak host plants at any time, and that is what actually happens. Thus we find that although the percentage germination is similar in the case of untreated and dehusked seed and treated and husked seed, the percentage infection in the case of the former is 5% greater.

Then following the same horizontal columns to the right we see that the percentage germination is the same in the case of dehusked and treated seed sown flat and dehusked and untreated seed sown upright and almost at soil level. Here, too, a similar explanation can be given. In the first case the host /

host is given a start by removing the husk and stimulating it with Ceresan and the fungus is poisoned. In the latter case a similar start is given by placing the embryo almost at soil level, so that it has no soil to push through, and the mycelium is exposed to atmospheric dessication. Since the results of the poisoning and mere dessication, which is never complete, are not similar on the fungus we find that the percentage infection is double in the case of the seeds where the latter condition (dessication) obtains.

In cases where the seed is stimulated and given the least resistance for the coleoptile to grow up, and the fungus poisoned, striking results are to hand. Almost cent per cent germination and 98 % healthy plants are obtained. Considering the comparatively high (40%) percentage of infection of the seed employed in the test the results are so promising that nothing more could be expected.

The function of the Ceresan is to poison the fungus and stimulate the host plant. This is experimentally proved by sowing treated and untreated seeds in soil and on filter papers. On filter paper no mycelium comes out of the treated seeds, while the untreated seeds are overgrown by the fungus. On filter papers, however, the host is also poisoned to a certain extent and the root production is inhibited greatly. In soil the poisonous effect is not present and the plant develops normally and its growth is slightly stimulated perhaps by the mercury salts present in Ceresan. The latter substance /

substance also poisons the fungus in the soil. It is very difficult to say what constituent of Ceresan is responsible for the results. As the effect of Ceresan on oats in soil is goverened by certain biochemical laws and the consideration of these is out of the scope of this paper, we will refer to the constituent as the 'active' constituent. This 'active' constituent dissolves in water held by the soil particles and forms a protective film round the grain.This solution also passes into the cavity formed by the husk and the grain proper from the upper end and thus is effective even in the husked seed. Since it is almost impossible to cover every seed with the required amount of Ceresan when this is used commercially on a large scale, certain seeds remain uncovered in the treated sample and thus give rise to diseased plants. However, if a thorough mixing is ensured all seeds are covered uniformly for all practical purposes. It may be noted here that large amounts of Ceresan are poisonous to the host as well, and thus should be avoided. Ceresan is only effective in the right concentration, this being about 2 ounces to a bushel of seed, and when thoroughly and evenly mixed.

As pointed out before Ceresan is only effective in those cases where the seed is only infected in the pericarp or the husk. In cases where the infection reaches the endosperm or embryo it is useless, as the embryo is killed right out before Ceresan can have any effect on the mycelium. In the case of /

of an infected endosperm it is possible for a seedling to develop from the seed but the 'crown' in such cases is badly attacked due to internal infection. Under good cultural conditions the plant may send out secondary roots from the lowest node of the stem and thus recover. But such cases are very rare. Ceresan has been used largely during the last three years in Scotland but in some cases its methods of application have been so unscientific that rather poor results were obtained by farmers. Succesful experiments have, however, been carried out at various places in Scotland and the use of Ceresan largely recommended. Unfortunately the actual importance of Ceresan has been too much exaggerated.

The only control which can be suggested at this stage is the selection of disease-free seed and shallow sowing. Slightly infected seeds should be treated with Ceresan, and the treatment should be carried out carefully. Sowing the seed upright and removing its husk are advantageous, but unfortunately it cannot be carried out under field conditions. Good cultural conditions and manuring will certainly minimise the disease, as the active growing plants will be able to escape the disease. Chile salt peter has been found to be effective in controlling the disease in Germany, but the evidence is only circumstantial and no definite experiments have been carried out. It is worthwhile trying this substance under Scotxish conditions. Various disinfectants have been suggested for seed treatment, but Ceresan seems to be the best.

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General Discussion.

The method of the infection of the embryo by the mycelium needs a little further discussion than that already given. Considering the two recent papers on the subject we find contradictory statements as regards the penetration of the mycelium into the host, and also the very source of this mycelium. Rathschlag states that he was unable to find the mycelium of Helminthosporium in the Pericarp, Testa, Endosperm, Scutellum and the Embryo of the seed in any case. He only found the mycelium and the spores in between the grain and the husk and on the outside of the grain respectively. I have found the mycelium in all parts of the grain, the extent of penetration depending upon the time and severity of the attack. I do not see any reason why he failed to observe the mycelium in the parts mentioned. Probably he was dealing with a sample of seed which was infected rather late while the crop was standing, and the fungus had very little chance of invading the internal tissues before the harvest.

Turner and Millard found the mycelium in various parts of the grain and have stressed the importance of this mycelium in the pericarp as a source of infection. They have made certain other statements which I have confirmed during my work. Unfortunately they have left me an oppertunity to differ from them in the actual penetration of the mycelium and its distribution in the various parts of the host. They start to explain /

explain the penetration of the host when the coleoptile and primary roots in their normal growth must have reached a length of more than one centimetre. This is a stage reached within eleven days after sowing and I do not see any justification in calling the germinated embryo, which has given rise to a seedling, still an embryo. They describe the first attack of the so called embryo at the junction of the scutellum and the epiblast, and then proceed on to say that the mycelium travels up the cells of the coleoptile, and inwards to the rudimentry coronal roots, and downwards to the seminal roots. Then further they say 'the critical region for the infection lies, however, in the cells of the coleoptile where the passage of the hyphae through their wide lumen is comparatively easy'. I do not mean to say that the fungus cannot enter at the points mentioned and does not travel in the directions stated, and I agree that the penetration of the coleoptile through the cuticle is difficult and slow at the time mentioned, but I do not see any reason why the mycelium should not penetrate the coleoptile and the coleorhiza just after they have started activity and have thin-walled and functionally undifferentiated cells.

The mycelium resumes activity much earlier than the embryo and penetrates inwards and outwards from the seed coat at all places, especially at the embryonic end, and all parts are susceptible to the attack when the embryo has just resumed its active growth. I quite agree that the points of junction of the scutellum and the epiblast may be the only weak /

weak points where penetration can succesfully occur into a seedling eleven days old, but this does not mean that the coleoptile cannot be infected directly with success earlier than eleven days. I find that the infection of the embryo can take place at all points when it is at a stage of susceptibility; this being the time from the resumption of the activity till the coleoptile is about 1.c.m. long and still enclosed in the husk. Upto this stage the young plant may safely be described in the embryonic stage, though not in the strictest sense of the word,but after this it is no longer an embryo, but a seedling, and the infection of the seedling may or may not be succesful, as they also describe.

I agree with them that the leaves are infected from the coleoptile by the inward penetration of the mycelium as also described by Smith for H.gramineum, but Turner and Millard think that they are unable to agree with Smith. I find a close agreement in their statements which are cited below:-

Turner and Millard state ' the fungal threads move (from the coleoptile) towards the rolled primary leaves and penetrate the first leaf on its outer edge. Meanwhile the leaf is elongating and the cells are being withdrawn from the region of infection so that the lesions develop as small scattered areas, lying in rows which may later join to produce the stripe form. When there is extensive infection in the coleoptile cells and when growth is delayed, the hyphae will pass through the first leaf into the second and third--'.

Smith states 'Thus, when hyphae break through the inner epidermis (of the coleoptile) and come into contact with the first leaf, penetrations are likely to occur on a longitudinal strip or strips, corresponding to those on the coleoptile. The upward growth of the leaf will also brush a vertical strip against the externally applied mycelium. The inrolled part of the leaf will escape penetration at this time, as will a greater or less proportion of the exposed surface, owing to the variable amount of mycelium which will have traversed the coleoptile'.

Turner and Millard are under the impression that under normal conditions only the first leaf is infected and the infection of the second and third is abnormal, but according to Smith the infection of the following leaves is a normal phenomenon. They agree, however, that the primary infection stops at the third or fourth leaf stage, which I also confirm. It may also be stated here that the infection may even stop at the first leaf stage and the second and the third leaves might emerge uninfected if the conditions for the parasite are unfavourable. As for their views on the penetrations let their statements speak for themselves. To me it appears that there is no difference in their views in general, and that is what should be expected under the conditions when the facts on which the views are based are similar, and I beleive that the explanation given by Smith for H. gramineum holds good for the 'primary stripe' form of the disease of oats. /

I do not find any indication that the fungus can live in a semi-symbiotic condition in the growing point of the plant like the smuts. The growing point, when attacked, fails to perform its function of producing new normal parts and eventually dies. The fungus then spreads all over the plant and the result is that the entire plant dies in a short time. I am also unable to confirm Ravn's views that H. avenae behaves like smuts in its pathogenicity.

The symptoms of the disease as described by me in this paper are typical and all sorts of variations are possible. The stripe form which is obtained by the external application of the mycelium on to the inrolled leaf can have many variations. The stripe may be continuous or discontinuous. The various parts of the stripe may be long or short, but they can never be called spots; Turner and Millard actually call them areas which I think cannot be interpreted as spots as they have done in their paper. The possible reason why we get the discontinuous stripe is the change of conditions from comparatively warmer weather during the day to a low temperature at night. The mycelium grows actively during the day and attacks the various parts in contact with great rapidity, while at night its growth is retarded and in certain cases the fungus is actually stunned and cannot resume activity for several hours even when brought to normal conditions, and thus fails to infect the part in contact. /

The time taken by a leaf to emerge from the enclosing leaf sheath is very short, and under normal conditions of growth a leaf fully unfolds itself within three days.The growth of the host is fairly rapid and is not affected by the cool night, and thus the various parts in contact with mycelium at night escape infection. From this it does not follow that a certain part of the plant kept in contact with the mycelium at low temperatures will not be infected. It certainly will be infected, but only when kept for a comparatively longer time, and the actual infection becomes visible rather late. Thus the part between the two separated portions of the stripe may be infected and yet the actual signs of infection absent. Thus I see no justification in calling the various parts of a stripe merely spots. The stripe is really complete but apparently divided into scattered areas as described by Turner and Millard. These apparently scattered areas have been interpreted as spots by them and the stripe is supposed to arise as a result of their union, although the stripe in its real form is already present.

As for the primary spot form of the disease I do not propose to suggest any definite theory at present, because the actual course of the hyphae in the leaves was not investigated. A probable explanation, however, can be given from the actual behaviour of the fungus and the structure of the leaf. The fungus grows up the leaf apparently invisible, and /

and makes itself conspicuous near the tip. The reason for such a supposition is that the fungus has been isolated from various parts below the apparent point of infection, and the first two or three leaves have hydathodes near their tips. These hydathodes are surrounded by an area of losely packed cells which retain an abundance of moisture and thus are more favourable for the growth of the fungus. Here the fungus grows luxuriantly and makes itself conspicuous in the form of a spot. From this place it gains a free entry into the various tissues of the leaf, this entry being denied during its upward growth by the mechanical resistance of the thicker-walled elements. The result of the downward invasion is the death of the leaf.

The secondary phase of the disease is quite independant of the primary attack as stated in the earlier part of this work. This is brought about by the spores which are dissiminated by wind and rain. These spores are formed on the dead leaves, the victims of the primary attack.

As regards my justification in dividing the species into two sub-species I have mentioned the two chief factors which draw a line of distinction, and the weight of the evidence speaks for itself. The difference between the two sub-species is in a way more remarkable than the difference between the former H.avenae and H.teres, but I do not feel justified in raising my two sub-species to a full specific rank.

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FUSARIUM.

It is a well known fact that the cereal crops are susceptible to various species of Fusarium, and that as a result of this attack considerable losses are experienced every year in most of the cereal growing countries. A short review of the literature is desirable here, owing to the fact that much work has been done in this direction and we have a fairly clear conception of the importance of Fusarium as a parasite of cereals. No attempt is here made to collect all the existing literature on the subject as a good review of the old literature has been given by Atanasoff in his monograph. Only a few important papers are considered here so as to have a general idea of the existing literature and the present position of the problem.

Much interest was shown in the pathogenicity of Fusarium towards cereals by Schaffnit in Germany who published many papers on this subject in 1912 and thus placed the question of the pathogenicity of Fusarium (towards cereals) amongst various other problems of scientific interest. At the same time Naumoff produced various interesting accounts of the'intoxicating bread' in Russia and pointed out that this trouble was due to the presence of Fusarium roseum (Gibberella saubinetii) and F.subulatum (F.herbarum v. avenaceum) on the infected grains. The two Fusarium species are alleged to produce red or pink sporodochia on the infected grains and sometimes perithecia may also be seen. /

He emphasizes that the presence or absence of these fruiting bodies is not the only conclusive test for the presence or absence of these fungi. The mycelium of these fungi may be found in the seed tissues and this mycelium regains activity under favourable conditions. He demonstrated the presence of the fungus mycelium in all the tissues of the stem except vascular bundles and the primary meristem, and that this mycelium is both intra- and inter-cellular. It may be noted here that I have observed the mycelium in all parts of the stem without exception. In fact the presence of the fungus mycelium in the vascular bundles is responsible for the seedling blight and deaf ear stage of the disease caused by Fusarium. He gives various temperatures suitable for the production of the perithecial stage of Gibberella Saubinetii and destruction of the mycelium of two species of Fusarium in the seed tissues without any injury to the embryo. Much interest was taken in this work owing to the fact that the toxicity of these organisms had been previously demonstarted by Gabrilovitch who made hypodermic injections into frogs with the extract of the fungus from a pure culture. The frogs died within four hours owing to the toxic effect of a nitrogenous glucoside supposed to be present in the fungal extract.

In human beings the effect of poisoning as described by Gibrilovitch are headache, general weakness and frequent vomiting. It may be of some interest to mention here that I swallowed a culture of Fusarium herbarum v. avenaceum in order to see /

see its effect on me. This action was prompted because of the denial of the toxicity of Fusarium infected grain by some workers on Fusarium. The symptoms as stated above are fairly correct and I experienced them all except that I had only one vomiting fit and a long sleep the next day. Later, however, I had to take a purgative to clear up my system and get rid of the prevailing diziness.

Then came the work of Atanasoff in 1920 from U.S.A. He described in this paper the common cause of the wheat scab so prevelent in the United States, and his work may be taken as the first of its kind and a revelation to Europeans of the real importance of Gibberella Saubinetii in relation to cereals. In Europe the different stages of the disease caused by G.Saubinetii were regarded as absolutely independant and non-related, and that different organisms were responsible for each.

Two other important works on Fusarium by Wollenweber and Sherbakoff appeared, but these works did not deal with cereals. They dealt with Fusaria of sweet potatoes and potatoes respectively. They described the organisms in hand with extraordinary ability and care, but I am of opinion that at that time they did not realise the shallowness of the characters on which they separated the various species. They found differences between various organisms as would naturally be expected and without sufficient consideration of the value and validity of differences raised such organisms to specific /

specific rank and thus increased the number of the already confusing multiplicity of species. The characters such as breadth, length and shape of macroconidia and the presence or absence of other types of spores which decide the line of dèmarcation between two closely related species are so mobile and unimportant that to rely upon these characters for purposes of classification is a folly. Since the works of Brown and others this mistake has been realised and Wollenweber in his latest edition of the Fusarium monograph published in 1931 has corrected this mistake and cleared the ground considerably. Still there remains much to be said about Wollenweber's latest work. These works as stated though not actually dealing with the cereals, are of considerable importance in this direction. They describe some organisms that are responsible for cereal diseases, and their works are of an indispensible aid to a worker on Fusarium.

In 1923 two very interesting works on the Fusarium problem were published. The first by Atahasoff deals with the Fusaria of cereals and the second by Morris and Grace Nutting is about the species of Fusarium on potato tubers. Atanasoff has taken to the pathological side of the problem more than the actual systematic position of the causal organisms, While Morris and Nutting deal exclusively with the identification of the species of Fusarium found on potato tubers and discuss the relative importance of various characters used in taxonomy.

Atanasoff made a thorough study of the existing literature on the question of the pathogenicity of Fusarium on cereals and his review is complete up to the date of publication of his paper. He has appended a long bibiliography on the subject and in fact, to trace the history of Fusarium in relation to cereals would be merely a repetition of what Atanasoff has written. In this monograph he brings home to the workers on Fusarium the importance of the disease caused by its various species and points out all the stages of the disease, some of which had previously been overlooked by European workers. The 'Head Blight' stage of the disease was recognised for the first time as of real importance in considerably affecting the yield of crops. This stage, though noticed by some of the earlier workers on Fusarium in Europe, had rarely been ascribed to Fusarium. Amongst the various stages of the disease caused by species of Fusarium he mentions 'Foot Rot', 'Root Rot', 'Stalk Rot', 'Node Rot', 'Seedling Blight', and 'Head Blight'. He gives a description of ten species of Fusarium, two of them having perfect stages. He also discusses the various stages of the disease in detail and accounts for certain pathological symptoms.

He observes that ' whether the cereal crops are attacked in one or several ways by Calonectria graminicola (F.nivale or F. minimum) common in Germany and the Scandinavian countries; by Gibberella Saubinetii common throughout the wheat growing /

growing sections of the United States, Holland, Bulgaria, and Southern Russia; by Fusarium avenaceum common in northern Russia, or by F. culmorum, common in Holland, Germany, Sweden, France and Oregon U.S.A., or by numerous other Fusarium species , the Symptoms and pathologic affects are identical in all cases, under all conditions and in all countries'. Considering that the statement is a general one it is to be regarded as correct and to the point. There always remains the fact that all species of Fusarium are not equally aggressive and thus the variations in the symptoms whereever observed are of a quantitative and not a qualitative nature.

He recognises two distinct phases of the disease of cereals caused by Fusarium sp.. The first is an attack on the underground portion of the plant and the second an attack on the aerial parts of a fully grown plant. The first attack results in 'Seedling Blight', 'Foot Rot', and 'Root Rot', while the second results in spotting of the leaves and the blighting of heads. The rotting of the nodes may also be present in the case of rye, wheat and barley. 'In all cases the various attacks on the same host are entirely independent of each other'. A plant may be attacked in any part or several parts at a time, 'but in all cases these infections are entirely independent one of the other and may be caused by the same or different Fusarium species'. The word 'independent' needs a little explanation in both these statements. /

As he observes 'the infection of the roots does not presuppose the infection of the head or node, or vice versa' and that the infection from one centre does not spread to another, that is to say, the fungus mycelium does not travel up the stem from the roots to the head and cause head blight, or travel down the stem to cause root rot. In this sense the two phases of the disease are entirely independent of each other. But all the phases of the disease are interdependent indirectly, since in the entire absence of primary infection at the seedling stage over a wide area, the secondary infection, which is the cause of head blight, is very improbable. It does not necessarily mean that the infection of the heads is bound to follow as a result of an infection at the seedling stage which results in seedling blight and foot rot. The inoculum (spores) for the head infection comes from the primary stage of infection, and since the disease is seed borne, the inoculum for the primary infection is provided as a result of an infection of the heads. In this way each phase of the disease is dependent one on the other outside the host, but, within the host they are independent of each other. This is what Atanasoff observed himself and the above is merely an explanation given to clear up his views on the subject.

Atanasoff has discussed in detail the various phases of the disease and has given accounts of the symptoms and reasons for /

for their appearance. In general his work, so far as it goes, is correct, and I do not see any reason to differ from him; but he has omitted a very important phase of the disease as also pointed out by Bennett in his paper which appeared in 1928. The 'Deaf Ear' phase which Atahasoff has omitted or confused with 'Head Blight' is absolutely different from the 'Head Blight'. By deaf ears we mean ears devoid of grain, and this pathologic effect is brought about by the infection of the crown and roots of a fully grown plant and not the head only as in the case of 'Head Blight'. In certain cases the infection of the head may result in the total absence of the grain, but such cases are very rare, and, when present, cannot be distinguished from 'Deaf Ears' where the infection of the crown has also occured. Atanasoff has discussed three stages of the blighted grain that results from head blight, but in all cases he mentions the formation of the grain, whether the infection of the head occurs during or any time after the flowering period. The earlier the head infection occurs the smaller the grain remains. He has not mentioned any case where the ears may remain grainless. To me it appears that Atanasoff has overlooked the 'Deaf Ear' stage entirely and has not confused it with 'Head Blight'. In 'Head Blight' the grain is always formed but remains small, shrivelled and light in weight. In 'Deaf Ears', the ear becomes bleached and the grain is not formed at all, although the ears seem normal to all appearance. /

Such heads give an appearance of premature ripening similar to that given by the blighted heads. The 'Head Blight' is always caused by the infection of the head during or after the fertilisation of the ovary and the blighting of the head may be complete or partial. Head blight is independent of 'Foot Rot' and 'Root Rot' and can be seen in fields where the latter two phases of the disease are entirely absent. The 'Deaf Ear' phase results from the infection of the crown and the roots immediately after shooting the ear and the actual infection of the head may or may not occur. When the infection of the heads does occur it is not responsible for the pathologic effect on the ear. As a result of an attack of the plant at soil level the vascular bundles of the stem in the region of the basal node are clogged up by the fungus mycelium and thus the water supply of the aerial parts is cut off. The aerial parts,especially ears which have been developing normally till flowering time,dry up and result in 'Deaf Ears'. The infection of the crown and the roots after flowering period does not result in deaf ears but 'White Heads' are formed. These white heads are similar to the blighted heads in appearance, but here again they are produced absolutely differently. This much discussion will suffice at this stage and more will be said later while discussing these phases of the disease in the present account.

Morris and Nutting have reviewed the literature in general giving their own views on the classification of the genus Fusarium. /

They have pointed out that certain species are merely synonyms of each other and it is doubtful whether any great difference exists between certain closely allied species. To quote their own words'it is doubtful whether there is enough difference between the species F.subpallidum, F. subpallidum v. roseum, F. clavatum, and F. discolor to warrant more than one species'. This view has partially been taken by Wollenweber who has placed these species under Fusarium sambucinum and reduced them to the rank of a form, with one exception where F. clavatum has been provisionally placed under F. flocciferum. The literature in their paper and the above monograph is so finely reviewed that I feel it unnecessary to repeat the whole thing again.

After the appearance of these two works, a Fusarium conference was held in America and the chief workers on Fusarium were invited to attend. At this conference the fundamentals for the classification of the genus Fusarium were laid down and a key for running down the various sections of Fusarium was drawn up. This work standardised the sectional classification but the classification into species remained unsettled. Many organisms not worthy of their names enjoyed specific names in those days of darkness.

In 1927 Reinking and Wollenweber published a detailed account of the tropical Fusaria numbering forty eight in all. Out of this large number as described by the authors there are seven new species, six varieties and one form. This work has /

has an outstanding feature in that it deals with fusaria of vastly varied affinities. The descriptions of the details are excellent but here again the same mistake of splitting up species into varities and forms has been made and many useless names involved. There is nothing in this paper concerning cereals so a critical review is unnecessary.

In 1928 came two papers, one by Bennett from England and the other by Simmonds from Canada. Bennett deals with F.culmorum and F. avenaceum. He is the first English worker to establish the pathogenicity of these two species of Fusarium and to make a detailed study of conditions favourable for the disease. He gives descriptions of the two organisms which he obtained from British cereals and also discusses the various stages of the disease. He recognised the 'Deaf Ear' stage and has given it a place of due importance with little or no exaggeration. He suggests certain control measures merely from the study of existing literature, although he himself did not do experimental work in this direction. In general he confirms the views of Atanasoff. He has taken a few isolated lines from Atanasoff's work and made criticisms on them which I think is not justifiable. I have gone through both these works very carefully and find a very close agreement in their views, the only difference being that of expression. Bennett is absolutely right in criticising Atanasoff when the latter states that the European workers failed to see the 'Head Blight' stage. The head blight stage had been recognised in Europe but due importance had not /

not been given. Attanasoff has laid more stress on the head blight stage, while Bennett considers deaf ear stage to be of more importance(this latter stage had either been overlooked or confused with 'Head Blight' by Attanasoff).

Simmonds discusses the pathogenicity of F.culmorum towards oats and gives details of invasion of the host at various stages. He discusses the anatomy of the young oat seedling and traces the course of the fungus in the host. He also gives cultural characters of the organism and points out that comparatively high temperatures are suited to the fungus. He tried many seed treatments for the control of the disease and found that some of them such as Semesan,Germisan, Uspulum,Tillantin, Segetan, Urania and others were fairly effective, Formalin and Sulphur did not control the disease. He however does not recommend any of the treatments for general use. He also carried out certain experiments to find out any resistant varities of oats, but the results were mostly negative. Only one variety (Joanette) of black oats gave some promise of partial resistance. He has also appended a long bibiliography on the subject.

After this appeared three more papers by Bennett. In these papers he describes Gibberella Saubinetii and F. scirpi and establishes their pathogenicity on the cereals. G.Saubinetii is described in two papers, the first one deals with the morphology and the other with the physiology and pathogenicity of the fungus in question. His third paper on F.scirpi is also of the same type. These papers are not of much value as /

as compared with his first paper of 1928 as the fungi described are not prevalent in Britain. The temperature requirements of these fungi do not allow them to be of any economic importance in this country, as they require relatively high temperatures. F.scirpi is not a very serious parasite. The critical review of these papers is not needed owing to the fact that in general these resemble the first paper by the same author.

The rest of the papers by Leonian, Coons and Strong, Brown and Horne, Horne and Mitter, Mitter, Chih Tu and others all deal with the phenomenon of variability in the genus Fusarium and its classification. These papers will be considered while discussing the phenomenon of variability.

The latest monograph of Fusarium by Wollenweber which appeared in 1931 is remarkable in that a large number of species have been amalgamated and many reduced to varities. Certain new forms have been created in this grouping of the species. Though the axe has worked a lot there still remains a great deal of useless shrubbery that must be removed. However this is the first advance towards a clear conception of the species and in due time we hope to get further contributions so that definite lines of demarcation between species be drawn. This work is worthy of praise and workers on the Fusarium problem should be thankful to Dr. Wollenweber for his untiring efforts to clear up the muddled situation of the taxonomy of the genus Fusarium.

At present the pathological side of the problem is by no means clear and we have to learn a lot about Fusarium before we can have a true conception of its behaviour. The physiological side also needs further examination and the phenomena of variability and mutation are worthy of consideration. At present we know very little about the conditions which govern these two phenomena and it yet remains to be proved whether the mutants and variants are of any real importance in the taxonomy and pathogenicity of the species of Fusarium. A considerable amount of evidence is accimulating on this problem and it is claimed that the variants and mutants really have a great bearing on the pathogenicity of many species of Fusarium. This question forms a part of the work and more will be said about it later in its proper place.

Geographic Distribution.

Species of Fusarium that are responsible for diseases of cereals are distributed all over the world where ever cereals are grown. The relative importance of any disease caused by these fungi, however, depends upon the quantity of the fungus material, species of Fusarium concerned and the climatic conditions of the country. Different species of Fusarium have different temperature requirements for their favourable growth and this factor coupled with moisture content of the atmosphere marks the limit of distribution of various species. Certain species of Fusarium may be found existing under unfavourable conditions in some part of the universe, but their importance as parasites is neglegible.

Fusarium nivale (Calonectria graminicola) is found in the northern countries of Europe and there it assumes the role of a serious parasite of cereals causing a disease commonly known as the 'Snow-mould'. In southern parts of Europe though F. nivale has been recorded no information is available as to its importance as a serious parasite of cereals. In Britain this fungus is not known to occur in the south, but it has been recorded by Millard on lawn grass in Scotland and by Bennett on cereals in north of England. I have found F. nivale causing a serious disease of oats in Scotland and I have isolated it from widely different fields and seed samples.This fungus requires a low temperature for the production of /

of its conidial and the perithecial stage (Calonectria gram minicola) and thus its geographic distribution is limited to the cold regions, especially to northern Europe. An other reason why F. nivale is more prevalent in the northern states of Europe is that the principal cereal crop in these countries is rye and this cereal is especially susceptible to this fungus.

Gibberella Saubinetii on the other hand requires a comparatively high temperature for its growth and the production of its conidial stage as well as the perfect stage. The conidial stage of G. Saubinetii (F. graminearum) is produced abundantly under very moist conditions, while the perithecial stage does not require much moisture. So we find in nature that the conidial stage of G.Saubinetii is not so abundant as its perithecial stage. With these temperature and moisture requirements this fungus is abundantly found in the U.S.A. and Southern Russia. Another factor that counts for its abundance in these two countries is that the principal cereal crop there is wheat and this cereal is more susceptible to G.Saubinetii than any other cereal. The sowing of wheat after maize in the same fields in these two countries is also responsible for its persistance, as maize is also attacked by G. Saubinetii.

Fusarium culmorum also requires a high temperature and thus is not a very serious papasite in northern European countries although it is prevalent in the southern states of Europe. Various /

Various strains of F. herbarum have different temperature requirements and thus the distribution of this species is not limited to any part. Its ability to attack all cereals alike, goes in its favour and thus this species of Fusarium is more or less cosmopolitan and its importance as a parasite is far greater than any other species.

Different species of Fusarium causing disease are reported from the following countries; Britain, Holland, Dehmark, France, Germany, Poland, Czecho-Slovakia, Austria, Italy, Switzerland, Russia, Norway, Sweden, U.S.A., Canada, Australia, South Africa, New Zealand, India and Japan. Records from other countries are not available although species of Fusarium are undoubtedly present in other countries and cause serious losses as in the above mentioned countries.

Losses experienced in almost all countries are quite appreciable, but the actual figures are not available from any country. Atanasoff has given some figures from the United States of America but these figures are by no means complete estimates of the real losses experienced every year due to the Fusarium attack of the various cereals.

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Sources of Infection.

Conidia, Ascospores, Chlamydospores, Mycelium and Sclerotia are the possible forms in which a species of Fusarium with a perfect stage in its life-history can be disseminated from place to place and cause infection. In the case of species without a perfect stage the conidia, mycelium and sometimes the chlamydospores are the only forms in which these fungi can be spread. The chief sources of these spore forms and the mycelium are the diseased grains used as seed and the infected stubble in the soil. Some species can continue to live in the soil as saprophytic forms and when any cereal crop is sown in such a soil it becomes infected from the mycelium in the soil.The relative value of each source and the importance of each form in which the fungus over-winters and is disseminated may be considered separately.

Seed:- The seed obtained from a diseased crop is infected to various degrees depending upon the conditions under which the crop reached maturity. The infection of the seed may be in the form of mycelium in the seed tissues or in the form of spores on its outside. Sclerotia and perithecia are of rare occurance on the seed and when present the condition of the infected grain is such that it is incapable of germination. The most important form in which the fungus perennates is the mycelium in the seed tissues. The extent of the invasion of the kernels depends upon the time at which the /

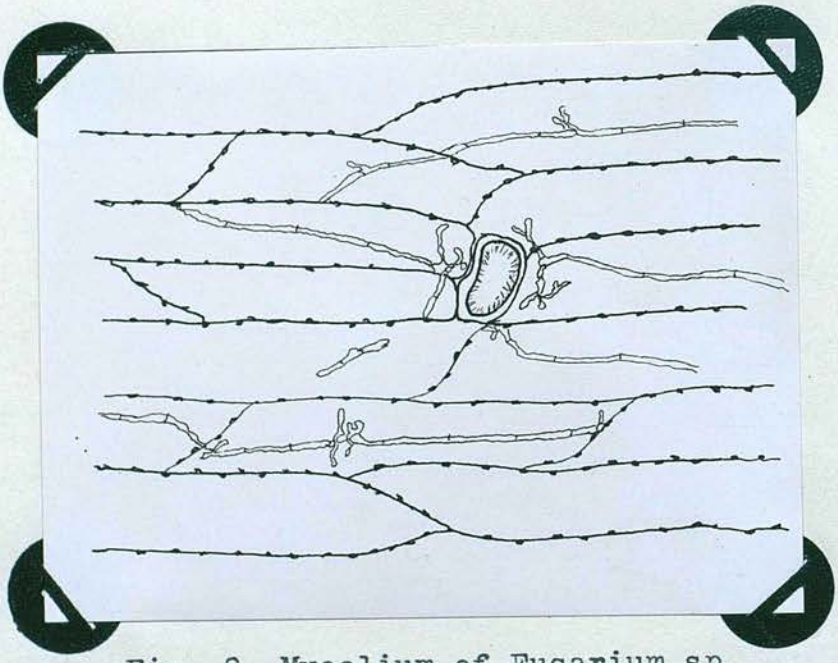

Fig. 8. Mycelium of Fusarium sp. in the outer-most cells of the pericarp of an oat grain.

the head is attacked. In most cases only the seed coat is infected and the endosperm and the embryo are free from the fungus. In other cases whole of the grain is penetrated by the fungal hyphae and such grains, as a rule, fail to germinate. If,ever, such grains germinate they form badly diseased seedlings and these are the chief source of the fungus for the infection of the aerial parts. When seeds, infected only in the seed coats, are sown they also form diseased seedlings which sooner or later succumb to the attack of the fungus and later form the source of infection for the whole crop. The mycelium spreads all over the dead remains of the seedling and forms spores which are scattered by wind. In the case of forms having a perfect stage, perithecia also develop on the dead seedlings and the ascospores play a prominent part in the spread of the fungus in spring.

Stubble and Soil:- The fungus also spreads into the soil from the dead remains of the seed and lives as a saprophyte till the favourable conditions approach. When the crop is about two to three feet high the soil mycelium is sheltered from sun and winds and it grows out into the air and form spores. This soil mycelium and the mycelium in the dead remains of the seedlings and ungerminated grains also spreads on to straw and other organic matter which is always present in the fields. On straw this mycelium forms spores that are disseminated by wind and deposited on the aerial parts of the plants. The nodes of all cereals are more or less succulent and /

and are easily attacked by these spores and the plants break as a result of the rotting of these parts. The spores that are formed in the slimy masses are not spread by the wind as they are glued together. These spores are spread by rain and washed down the stem to the leaf sheaths and its base. They may also be splashed from one plant to another. When a drop of water is placed on such a slimy mass of spores, the latter are detached from the conidiophores with a violent force and begin to move freely in the drop of water. When these spores reach the crown of an uninfected plant they germinate and the resulting mycelium spreads on to the tissues of the crown. Certain portions of the crown decay without any obvious papasitic attack and such parts form the best medium where Fusarium species establish themselves. Aided by some saprophytic fungi and bacteria they destroy the whole of the crown in a short time. This infection of the crown in plants two to three feet high results in 'Deaf Ears','White Heads' and 'Thinning Out'. The roots of the plants are similarly attacked at any age and the entry of the fungus may be through the epidermis or the root hairs as suggested by Simmonds. The 'Root Rot' and 'Foot Rot' phases of the disease commonly result from such an attack of the grown up plants, although soil mycelium may also produce the same effect independently.

The aerial parts become infected by spores produced on the dead plant remains and under suitable conditions which vary for /

for every species, the disease tends to become epidemic in certain limited areas. The most important part of the plant that is infected in this manner is the head. Spores are deposited on the glumes by means of wind and there they germinate and penetrate the tissues of the glumes. If the conditions remain favourable for about ten days the fungus spreads over the whole ear and penetrates inwards, thereby involving the kernels. Adams is of opinion that in all cases the entry of the fungus into the seed takes place through the embryo where it establishes itself before it penetrates into the endosperm. This view has been discussed by Atanasoff and rightly discarded. Although the embryonic parts are easily attacked by the fungus it does not follow that these are the only places of entry for the fungus. I have observed that the fungus can and actually does penetrate the grain at any point and this point in many cases is marked by a slight discoloration on the seed coat. When the grains are attacked very young the testa and the aleurone layer do not act as barriers for the fungus and the penetration of the kernels is equally easy at all points. In almost mature grains the embryo is more easily invaded than any other part due to the fact that the aleurone layer opposite the embryo in all the cereal seeds is almost absent or broken into pieces. In mature grains this aleurone layer acts as a barrier at all other points and thus the mycelium can enter the embryo with great ease. Also there is more moisture retained by /

by the glumes at their bases than at the tops and the embryo lies in the lower part which is more favourable for the growth of the fungus. In this state, however, it is not possible for the fungus to spread into the endosperm as the Scutellum acts as a barrier on that side. So in almost mature grains the fungus can only penetrate the pericarp and other layers present between this and the aleurone layer. Such infected seeds form the source of the fungus for the future crop and thus the cycle is kept up from year to year. The mycelium in the seed tissues remain viable for atleast two years.

The spores of Fusarium present on the outside of the grains are rarely in a condition as to be of any importance in causing the disease. These spores remain not longer viable after a storage of the grain in a dry place for about six months. However, the species that form chlamydospores within the conidia can live in this form on the outside of the grain. In such cases these spores have the same importance as the mycelium in the seed tissues.

In general it might be concluded that the mycelium in the grain tissues is the chief form in which the fungus perennates and the spores produced on the dead seedlings are the chief form in which it is spread in a standing crop. Sclerotia and chlamydospores are of great assistance in over-wintering and perithecia also play a similarly important part.

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Description of the Disease.

It has been noted during the present study of the Fusarium problem that in general the pathologic effects produced on various cereals as a result of an attack by different species of Fusarium are identical in all cases, and thus the symptoms as witnessed are similar regardless of the species of the cereal and Fusarium concerned. However, it must be remembered that there are all sorts of deviations from the normal typical course of the disease and such variations cannot usefully be incorporated in a general statement as the above. Indeed the nature of the cereal and the pathogen determine to a great extent the fate of the crop, and as will be pointed out in the following pages these two factors along with the environmental conditions govern the course of the disease. Some species of Fusarium such as F. nivale (Calonectria graminicola) are more virulent at comparatively low temperatures and are more dangerous at the seedling stage than any other stage in the development of the host, while certain other species such as F. culmorum are more virulent at high temperatures and are more important in the head blight phase of the disease.

Again the various strains of the same species of Fusarium may not have the same virulence and thus certain quantitative differences might be observed in the symptoms exhibited by the host as a result of an attack by such different strains. However, the fact that all Fusarium species described /

described here can produce all the different phases of the disease, cannot be denied. Certain strains of a pathogenic species of Fusarium may be completely harmless and incapable of attacking the host under normal conditions, and in such cases no disease is produced at all. Such cases are very common and that is what should naturally be expected. After all, a species as we know it, is just a connected or coherent group of individuals related genetically, and all sorts of variations in the characters of these individuals are bound to be noticed in daily life. The same can be said of the species and individuals of the host.

The description of the various recognised phases of the disease is given here with an explanation of the cause and the effect. Following the cycle of the disease from the time the grain is sown, till it develops into a plant and bears the grain in return, the following phases of the disease may be observed in order; 'Pre-emergence Blight', 'Seedling Blight', 'Spring Yellows', 'Foot Rot', 'Root Rot', 'Crown' and 'Stalk Rot', 'Thinning Out', 'Deaf Ears', 'White Heads', 'Head Blight' and the 'Blighted grain'. Various other names for different phases of the disease are encountered in literature, but the list given above represents all the known phases. All these phases are described below one by one.

'Pre-emergence Blight' :- As already mentioned under the source of infection the seed and the soil are the two chief sources of the fungus. So, while considering the various phases it is assumed that the seed is infected in all cases and the /

the fungus may also be present in the soil. The degree of infection of the seed will be mentioned as required.

When an infected seed is sown in a suitable soil, it germinates like a normal seed provided that the embryo is not infected or killed. In most cases, if the embryo is infected the seed fails to germinate under normal conditions, although the embryo may not be dead before sowing. In certain cases, however, the embryo manages to regain activity, but it has a very short-lived career. By the time it reaches the soil surface it is overcome by the pathogenic fungus and is killed. In other cases the embryo may be free from infection before the seed is sown, but the endosperm and the various other parts of the grain may be heavily infected (Such cases are quite few). When such seeds are sown, they also meet the same fate as those which were infected in the embryo. The embryo germinates, but it is attacked so vigorously by the fungus mycelium at this stage that it fails to push through the soil and come to the surface. The mycelium from the infected grain grows into the soil and also enters the young embryonic parts from various places of the grain which are rotten by this time. The mycelium in the scutellum cuts off all the food supply of the young plant from the grain and thus renders it more susceptible to the mycelium in the soil. The soil mycelium by this time grows all round the young coleoptile and coleorhiza and under such conditions the young seedling falls an easy victim and is immidiately killed. In this /

this way all such germinated embryos fail to come up the soil and a poor stand of the crop results. All such cases where the seedlings fail to push up to the soil level come under the 'pre-emergence blight'.

The losses due to the pre-emergence blight cannot be estimated if the seed sample is not tested for %age germination on filter papers in the laboratory. Under the test conditions of the laboratory even the severely infected grains germinate and they are counted amongst the germinated grains. In the field these grains fail to germinate or when they germinate they fail to appear above the soil surface, and the difference between the percentage germinations obtained shows the percentage of pre-emergence blight.

Any seed borne pathogenic fungus of this type can cause pre-emergence blight provided that the conditions of the soil permit its normal growth. Species of Fusarium and Helminthosporium can grow in soil very luxuriantly, and thus when these fungi are present in a severely infected seed they cause the pre-emergence blight. No data regarding the losses due to this phase of the disease are available from the previous literature and it is indeed very difficult to estimate the exact amount of losses. In certain cases 5 to 10 % loss in the stand of the crop may easily be expected due to pre-emergence blight. This phase of the disease cannot easily be distinguished from pre-emergence blight caused by other /

other agencies unless the seed along with its killed little shoot is dug out of the ground and carefully examined.

This phase of the disease can be produced by the mycelium present in the soil, and also by artificial infection. In all cases the young seedling is attacked in the same way and ultimately killed.

'Seedling Blight':- This phase of the disease is very commonly observed in poorly drained soils. The infected seeds that are sown in such a soil germinate rather late and the resulting seedlings are always weak. This phase of the disease results from an infection of the young seedling before or after its emergence from the soil. The blight always results when the seedling is attacked at the crown, roots and the coleoptile, and the first foliage leaf is also infected from the latter. The mycelium grows out from the infected seed into the soil and attacks the young coleorhiza and the coleoptile. The first symptoms of the attack when it is made before the emergence of the coleoptile from the soil are rotting and browning of the coleoptile and the coleorhiza. The mycelium present in the soil also acts in the same manner. The first foliage leaf which is enclosed in the coleoptile becomes infected at this stage and it may fail to emerge if the infection is severe. The severity of infection is controlled by the moisture and temperature of the atmosphere and the soil, and the nature of the organism infecting the seedling. For example, Calonectria graminicola attacks more severely if the temperature is low and the atmosphere /

atmosphere comparatively dry. On the other hand certain strains of Fusarium herbarum are more virulent under comparatively high temperatures and abundance of moisture. In all cases, whatever the combination may be, the resulting symptoms, that is rotting and browning of the infected parts, are exhibited at the seedling stage.

The primary and coronal roots are attacked by the mycelium in the coleorhiza and the rotting of the tissues proceeds on from the infected area outwards to either side. All parts of the infected tissues are invaded by the fungus mycelium and the xylem vessels are clogged up. The first foliage leaf when attacked becomes yellowish and more or less bleached after a short time and the mycelium grows out of it in moist and calm weather and forms spores that are dessiminated by wind and rain to other plants.

The crown of the young seedling is infected from the remains of the grain and this along with the remains of the coleoptile and coleorhiza form the centre of the mycelium where it is in a position to attack any part. From these parts it spreads on to other parts and the rotting proceeds till the water and food supplies are cut off and no means of comunication remain between the aerial and the underground parts. The underground parts completely rot and the seedling succumbs to the attack and wilts. Death of the aerial parts soon follows and this stage of the disease is termed 'Seedling Blight'. Seedling blight results in poor stand and this phase of the disease is particularly important as it /

it gives more material of the pathogen for further menace to the crop.

Seedlings that escape death at this stage go on living if the conditions are favourable for their growth and even when infected badly at the roots develop new roots from the upper part of the crown and thus carry on normally. The second and third foliage leaves usually remain free from the fungus and thus the seedling may go on developing normally upto the earing time. Atanasoff states that if infected plants are transplanted in healthy and well drained soil in pots they recover from the attack and develop normal ears. It is possible that the plants can recover if the root system was damaged only, but if the first node of the culm is infected it is impossible to produce a healthy plant from an infected plant. If the first node or even the crown be infected the water supply of the aerial parts is permanently cut off and the formation of the new healthy roots is of no avail to the plant. In fact if the crown be completely destroyed the formation of the new roots is not possible.

Losses due to seedling blight have been variously investigated and it has been mentioned that upto 10 % losses may be experienced in certain years. However, complete data are not available and it is doubtful whether the above estimate is really correct.

'Spring Yellows':- This phase of the disease is only observed /

observed in those cereals which are sown in winter. Some of the diseased seedlings do not die in the early attacks at the seedling stage in winter, but go on living till spring time although diseased at the base. In winter the parasite is not very aggressive, but it goes on invading fresh tissues slowly but steadily during the whole of the cold period. In spring the fungus again becomes active and affects the seedlings badly. On the other hand the uninfected plants grow luxuriantly and form a distinct contrast with the yellowish diseased seedlings. The diseased seedlings are stunted and show withering of the leaf tips. The underground parts are badly injured and show rotting in various degrees. Such seedlings wilt rapidly with the advancing warm weather. These diseased seedlings are termed 'Spring Yellows' by the farmers. In warm and wet weather the disease is most severe, while in cool and comparatively dry weather it is checked.

Bennett recommends the common practice of top-dressing a crop with a nitrogenous fertiliser as it stimulates the production of new roots that may help the plant to reach maturity.

'Foot Rot' and 'Root Rot' :- These two phases of the disease cannot conveniently be discussed separately owing to their close relationship. These phases may be witnessed from the time the seedlings show up on the soil surface till the crop is harvested. Infection of the host at soil level and the infection of the roots can take place at any time during the development of the host. In most cases, however, the seedlings are /

are infected before the actual signs of disease are visible. This infection occurs from the infected seed or soil and the seedling may be diseased severely or lightly, depending on the degree of infection of the seed and the conditions under which the seedling lives.

In warm and wet weather the roots of the diseased seedlings turn brown and begin to rot. This rot spreads upwards and downwards from the point of infection till the whole of the root system is involved and the 'foot' of the plant is also infected. At this stage the disease may be checked if the weather conditions change. However in this state the seedlings cannot carry on their normal function as the water supply of the aerial parts is cut off and the plant wilts. When such plants are pulled out they break at the base as if cut with a knife. The roots do not come up with the plant when pulled and on digging out they are found to be completely rotted. All parts of the infected tissue are invaded by the fungus mycelium which is both intra- and inter-cellular.

If the disease is not very severe in the early part of the development of the host, the latter thrives on till it reaches at the age of shooting out the ear. By this time the weather becomes warm and the field is covered up with the foliage that allows very little evaporation of water from the soil. These are the conditions that are favourable for the parasite and it grows out from the dead remains of the /

infected leaves that fall down and the rotted part of the crown and attacks fresh tissues. In this way the root system is destroyed and the 'foot' of the plant weakened and the other phases dependent on these two conditions follow soon.

'Crown and Stalk Rot':- This phase is witnessed in very damp soils. The mycelium present in the soil or in the dead remains of the infected root system grows into the crown of the stem and the latter turns brownish as a result of this attack. From this place the mycelium spreads on to the first node of the stem and the latter also becomes discoloured. The nodes of the cereals are somewhat succulent and are susceptible to the attack of the Fusarium spores that may be blown and deposited on these parts. From these infected nodes the fungus may spread upwards and downwards and cause rot. This condition is witnessed more usually in rye fields infected by Calonectria graminicola.

'Thinning Out':- This name is applied to the phase of the disease in which the plants infected at the base break away before the wind and bend over normal plants. These broken plants dry up and no grain develops in their ears. In this way the stand of the crop is materially affected and the farmers term this phase as 'Thinning Out'. This is the direct outcome of foot rot and crown rot.

'Deaf Ears':- This pathologic effect is brought about as a result of an infection of the crown and the roots of a fully grown plant immediately after shooting the ears. The vascular bundles /

bundles of the host are clogged up at the point of infection and thus the water supply of the aerial parts is cut off. Owing to the lack of water the whole plant wilts and dries up. The ears that have been developing properly uptil this time develop no grain and thus they are called 'Deaf Ears'. The head may or may not be infected and when infected the infection is not responsible for the pathologic effect under consideration. As stated this phase of the disease is dependent upon 'foot rot' and 'root rot' and is entirely independent of 'Head Blight'. The deaf ears give an appearance of a prematurely ripe head and acquire the same shade of colour as a normal ripe head of the cereal. Such heads are observed on plants that break down later against the blasts of wind. The infection of the roots need not necessarily in all cases produce deaf ears. Some roots may not be infected and these may be able to carry on the plant to maturity. In other ~~cases~~ cases the stopage of water may be late and in such cases the ovaries that are fertilised develop small grains. These later cases cannot be considered under 'deaf ears', but they come under 'white heads'.

'White Heads':- If the infection of the crown and roots occurs two weaks or more after the flowering period the formation of the grain is not checked although the grain formed remains small and shrivelled. This grain may or may not be capable of germination, depending on the time at which the water supply of the aerial parts is cut off. Note that here too the /

the pathologic condition of the grain is not due to the infection of the head, but of the crown and the roots. If such grains germinate successfuly they should remain free from the disease at the seedling stage if the soil in which they are sown is healthy. If,however, the head is infected later the seedling may become diseased.

'Head Blight' and 'Blighted Grain':- This phase of the disease is perhaps the most important phase as a few days of favourable weather for the fungus seriously damage the crops. It can be witnessed even in the best fields that promise a very good crop two or three weeks before the ripening of the grain. Head blight is independent of all other phases of the disease in so far as it does not stand in relation to any other phase directly. However it is indirectly dependent on the other phases such as seedling blight for the inoculum. Spores produced on diseased plants are responsible for causing this disease and these can be blown from one end of the field to another or to various adjoining fields. It has been shown experimentally by some of the American workers that the spores of these fungi can be carried long distances by wind and deposited in suitable places where they can germinate. It is doubtful however whether these spores that are blown long distances really play any part in the destruction of the crops, as I have seen fields that are badly infected by Fusarium, while the adjoining fields free from such disease.

The head blight is the result of an infection of the head during or /

or after the flowering period till the maturation of the grain. This phase of the disease is controlled by weather to a far greater extent than any other phase. In warm and wet weather the disease is severe and the presence of a slow but steady wind is a great advantage in favour of the parasite. The spores are spread all over the field and if the weather remains favourable for about a week the whole of the crop may be destroyed. Atanasoff has mentioned cases in which the crops promised a very good yield at the flowering time, but by the time the crop was ripe scarcely a grain was seen in any of the heads and some fields had to be left unharvested. I have seen only two such cases where a part of the field was not worth harvesting and the remaining of the fields was by no means any good.

Under ordinary conditions usually a single spikelet is infected and the first sign of infection is the appearance of a water-soaked area at the point of infection. This water-soaked area gradually loses its green colour till the whole of the glume is bleached. From this part the discoloration spreads all over the spikelet. The mycelium travels down the stalk of the spikelet and reaches the rachis and from this place it spreads on to the neighbouring spikelets. In the case of oats the infection of the neighbouring spikelets is by means of the spores that are formed on the diseased spikelets. The infection may occur through the rachis but the distance to be travelled by the fungus is very great and by the time /

time it reaches the spikelet in question through the rachis the spores formed on the other spikelet have already reached it.

In wet weather large masses of spores (Sporodochia) are formed on the diseased parts of the head and these spores are splashed by rain and blown by wind and deposited on the undiseased heads. In this way the disease becomes almost epidemic within a short period, and all parts of the head are involved. It all depends upon weather, whether the whole head is diseased or only one or two spikelets alone are involved. In dry weather only a few spikelets become diseased and the spread of the fungus is kept in check. Under favourable conditions the fungus spreads on to the rachis and when this happens the whole head above the point of infection dries up owing to the shortage of food brought about by the fungus. The infected part, or in cases where the whole head is involved, the whole head, acquires the colour of a normal ripe head of the cereal.

Atanasoff states that any part of the head may be diseased and that part may be the upper, middle or lower. I have never come across any head in which the lower or middle part of the head as a whole be diseased and the upper part free from infection, or signs of disease. It is quite common to find heads in which only a few spikelets are infected in any part of the head, but these spikelets do not form the middle or the lower part as a whole. According to his own reasoning which I /

I also support, when a certain part of the head is infected the vascular bundles of the rachis are blocked up at the point of infection and the upper portions of the head dry up as a result of shortage of food and water. He, however, reasons that one or two bundles may remain free and go on supplying the upper part till it reaches maturity. This however is not possible. One or two vascular bundles cannot supply a whole part of the head. They may nourish one or two spikelets, but the normal supply of the part as a whole cannot be undertaken by two vascular bundles in place of scores of them.

As a result of head blight the grain remains small shrivelled and light in weight. Whatever the time of infection may be, the grain is always formed and the size and appearance of the grain depends upon the time at which the head is infected and the weather conditions during and after infection. Some sort of discoloration is observed on all the diseased grains, but I have never seen sporodochia on the grains as mentioned by Atanasoff. If the infection occurs during or shortly after the flowering period and the weather is warm and wet the fungus attacks the young grain which remains very small, brownish or greyish in colour, light in weight and shrivelled. Such grains as a rule are incapable of germination. If however the infection occurs two or three weeks after the flowering time the grain is a little better but has the same symptoms as the above. Such grains can germinate, but in the field these are the grains which are responsible for /

for pre-emergence blight. Lastly the head may be infected shortly before the complete maturation of the grain. Such grains are of the same size as the normal grain but lack the healthy look of it. They have small reddish-brown spots on the pericarp in the case of wheat and rye and on the palea in the case of barley and oats. These grains usually are the cause of disease in the future crops as they pass undetected in a healthy seed sample.

In this way the cycle of the disease is kept up and repeated from the grain to the grain in the next crop. Although it has been assumed in the preceding discussion that the origin of the disease is from the infected seed, it must be remembered that the fungus in the soil has also the same importance if cereals follow cereals every year, but in the system of rotation of crops it is not as important as the mycelium in the seed.

Methods of Investigation.

Isolation:- Species of Fusarium were isolated from various sources and the methods employed were almost similar in all cases with little variations as suited to the occasion. Infected grains, wilted seedlings and infected parts of mature plants usually furnished the required material. The methods employed for isolating these fungi from plant tissues and the grains were different and so they will be described separately.

(a). Grains:- The grains were first surface-sterilised by either dipping in alcohol for a minute and then burning away the excess of the liquid or dipping in a 2 % solution of mercuric chloride for about 15 minutes and subsequent washing with sterile water. The first method in which the grains were left charred and surface sterilised, the embryos were injured considerably and usually failed to germinate. Owing to the above mentioned defect in this method it was only applied in those cases where the germination capacity of the grain did not matter. The second method of surface sterilisation was more suitable as the germination power of the grain is not interfered with, and the grains germinate normally.

Such surface-sterilised grains were incubated on moist filter papers in sterile Petri dishes or were cut into small pieces with sterile scalpels and these pieces incubated on malt agar plates. The Petri dishes were kept at a temperature of 24°C. /

in an incubator during the winter months, and in the laberatory during summer. Fungus mycelium usually grew out from the infected grains within three or four days and it was transfered to culture tubes containing different media. The infected grains were kept and watered as required, till the spores of the fungi were formed on them. It was found advisable to acidify the malt agar plates with lactic acid in order to check the growth of bacteria.

(b). Plant tissues:- As stated the wilted seedlings and infected plant parts provided the material of these fungi. The wilted seedlings were carefully pulled out of the soil and washed with water so as to remove the soil from the roots. The aerial parts and the roots were then cut into small pieces and these pieces surface-sterilised by dipping in 1% mercuric chloride solution as in the case of grains, or in a weak solution of formalin in 50 % alcohol. The plant portions were then washed with sterile water and incubated on malt agar plates. The mycelium of Fusarium grew out from these infected parts and spread on to the culture plates. Portions of agar with the fungus mycelium were then transfered to culture tubes of different media. In certain cases the mycelium had to be replated on acidified malt agar plate so as to get rid of the bacterial contamination which persisted in certain cultures even after repeated sub-culturing.

Clean cultures were obtained in all cases, but they had to be sorted out later by taking single-spore cultures, as mixed cultures of Fusarium often pass as pure cultures.

Methods for obtaining single-spore cultures:-

All the species of Fusarium studied during this work form in culture the typical macro-conidia on the aerial mycelium or in sporodochia or in pionnotes. These spores as a rule are capable of germination as soon as they are detached from the mycelium or the conidiophores. Four methods were employed for obtaining single-spore cultures from these spores.

(a). A liberal quantity of the spores was taken on a sterile wire and transfered to a drop of sterile water on a sterile slide. The slides were sterilised before use by dipping them in methylated spirit and then burning off the spirit. The drop of water containing the spores was then spread by means of a loop all over the surface of the slide and the water allowed to dry. This slide was then examined under the low power of the microscope and clearly isolated spores picked up by means of a very fine platinum needle. The platinum needle was sterilised by heat before use and was repeatedly sterilised after the transference of every spore. Usually the spores that were picked up by this method were transfered on to plated malt agar and the plates incubated at a temperature of 24°C. In other cases the spores were transfered to drops of malt agar on sterile slides. These drops were re-examined under the microscope in order to make sure that only one spore has been transfered to each drop. Drops with more than one spore were rejected, while those containing single /

single spores were transfered to culture tubes by means of an agar spade.

This method was only applicable in the case of Gibberella Saubinetii and Fusarium culmorum, as the spores of these two fungi are large enough to be picked up by a needle under the low power of the microscope. Moreover these spores are not very long and are fairly broad and were not injured while being picked up. The spores of all other species are long and narrow and it was found very difficult to pick up these spores separately as they usually cling together in masses. Also ~~the spo~~ these spores broke very easily and thus this method could not be successfuly applied. This method was also applied in the case of Fusarium sambucinum with success.

(b). In this method a suspension of spores was made in sterile water and this suspension diluted till a single drop taken from it showed a few clearly isolated spores. Plates of malt agar were then poured and a drop of this suspension spread on the surface of this plate by means of a sterile loop. This plate was then examined under the microscope and the spores located. Discs of agar with single spores were then cut by means of a dummy objective which could be swung in the place of a real objective after the spore was located. Such discs of agar were then re-examined under the high power in order to make sure that each contained a single spore, and then transfered to culture tubes.

This method also has its defects and the chief difficulty with this /

this method is that of locating the spores on the surface of the agar. The agar surface is never so even as to allow of a single focus to be used throughout the examination of the plate, and while adjusting the focus, the surface of this transparent medium is easily lost. Otherwise this method is very accurate and single-spore cultures can be obtained with little or no bacterial contamination.

(c). In this method also the spores are suspended in sterile water and diluted to such a strength that a drop taken by a loop of wire shows only three or four clearly isolated spores. A drop from this suspension is then spread on an agar plate by means of a loop and incubated. The spores germinate within two days forming small colonies easily visible to an unaided eye if the plate be held against light. These colonies are lifted with bits of agar by means of a needle and the agar piece transfered to a culture tube. In certain cases it is advisable to allow these colonies to grow till they attain a circumference of about 4 to 5.m.ms. The transfers are then made from the tips of the hyphae instead of transferring the whole colony, and in this way bacterial contamination is avoided, as the tips of the hyphae are usually in advance of the bacterial growth.

(d). The fourth method consists in mixing the suspension of the spores with the melted agar medium before plating. The medium such as malt agar is melted in the tube and one drop of the diluted spore suspension added and the tube thoroughly /

thoroughly shaken so as to ensure complete mixing. This agar is then plated and incubated. It is advisable to have the medium as cool as possible without setting before the spores are mixed, as in hot medium the spores are killed. Spores incubated in this way germinate to give rise to isolated colonies which can be transfered as usual. This method is not very accurate as we are not sure whether the fungus colonies are derived from single spores or more than one spore. It is convenient, however to mark the position of single spores by means of India ink. This can be done by examining the plate upside down and locating the spores under the microscope while looking through the medium. The position of singly isolated spores is then marked on the glass Petri dish.

Cultures obtained by all these methods were sometimes contaminated with bacteria or some moulds and so, it was advisable to make a fairly large number of cultures at a time. All contaminated cultures were rejected and pure cultures marked and the similarity of all cultures derived from the same source and on the same medium served to ensure the purity of all cultures. With advancing practice the contamination became less and less till a time came when all the cultures prepared were free from any contamination. In detailed work only one single-spore culture was used and marked as standard. Before starting the detailed work single-spore cultures were again made from the pure culture by either of the methods described. Whereever contamination was suspected during /

during the work single-spore cultures were again made and the culture re-standardised. In certain cases the phenomena of variability and mutation complicated the problems very much and so the cultures exhibiting this phenomena were treated separately and attempts made to stabilise and fix the various variants and mutants. Detailed accounts of these processes will be given later.

Species of Fusarium grow well on almost all acid media; basic media, however, are not favourable for their growth. It has been observed that cultures of Fusarium produce different types of growth on different media and produce different colours. The growth of these organisms was greatly influenced by the temperature at which they were grown and the nature of the medium and inoculum had a marked effect on the resultant growth. Repeated transfers from sporodochia reduced the production of the aerial mycelium considerably and the production of sporodochia was also adversely affected in the case of those species which as a rule produce abundant aerial mycelium. Abundant production of the sporodochia was obtained if the inoculum be from the sporodochia but its amount very small. Transfers from the aerial mycelium produced abundant aerial mycelium in the subsequent cultures, but the production of sporodochia and the intensity of the colour, both in the aerial as well as the submerged mycelium, was adversely affected. In many cases repeated cultures from the aerial mycelium did not produce any sporodochia in the subsequent cultures. Sub-

cultures from /

from the submerged mycelium usually produced normal cultures provided that the temperature and the medium did not interfere with the production of the 'norm'.

It was found essential that cultures of Fusaria should be brought into a state known as the 'norm' before all the spore forms and the perfect stage could be obtained. This point has been stressed by Wollenweber and others and I am in complete agreement with their views. A normal culture produced all the desired spore forms on certain media such as oat agar, provided that all the rest of the conditions were favourable. In order to find out the normal state of the fungus, it had to be grown on different media and at different temperatures. The conditions under which the cultures produced the desired 'norm' were then noted and applied in future. Failure to get the culture in 'norm' meant failure to get all the stages in the life-history of the fungus. All attempts to produce the perithecial stage of Calonectria graminicola met with failure until the culture was brought into the 'norm'. The very first transfer from this 'norm' produced the perithecial stage on oat agar. It took about three months to get this fungus into the normal form and from this example it is easy to form an idea how difficult the problem is. When once a culture was in the 'norm' it could be kept in that state for a long time provided that the conditions under which it was grown did not favour the reversion of the fungus into the abnormal form.

As stated the conditions which govern the production of the 'norm' are the temperature, the nature of the medium, the nature of the inoculum and finally the nature of the organism itself. Different species of Fusarium require different temperatures for the production of the 'Norm' and since there are different strains in a single species with different temperature requirements it is not advisable to generalise on any conditions for the production of this state even for a single species.

Many media were employed during the cultural work on Fusaria but the best medium for general work appears to be oat agar. Malt agar, potato agar, potato-dextrose agar, rice meal agar, and some synthetic media were also of indispensible aid. All these media were prepared in the usual way and I think it is unnecessary to give the details of their preparation as they are in common use in all the mycological laboratories. Whereever any special medium is used its constituants and preparation are given in the proper place.

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The Causal Organisms.

Almost all the organisms described here have been studied by various workers under the same or different names and attempts have been made to describe these species of Fusarium more or less accurately from the morphological point of view. The majority of the authors have failed to understand the importance of the cultural characters and their bearing on the taxonomy of these fungi. That the species of Fusarium react remarkably towards the environment has been realised lately and striking evidence has been put forward by Brown and others that the physiological and cultural characters of the species of Fusarium are as important in ~~the~~ taxonomy as the morphological characters. The chief importance of the physiological and cultural characters of these organisms, however, lies in the study of their pathogenicity. Some strains of a certain species of Fusarium are parasitic while others are harmless. Morphologically these two sets of strains cannot be distinguished from one another and certain workers dealing with only one type of strains declare the species to be perfectly harmless, while others working with the other type declare it parasitic. Since the two types of strains are morphologically identical a severe controversy arises between the workers as to the pathogenicity of the organism. If the cultural characters of these organisms are studied, the two types of strains are easily distinguished. Moreover some physiologic forms may also be differentiated within /

within the same race if the physiological characters are studied. In this manner we realise the existance of various physiologic forms, strains or races, biotypes and varieties within the same species and thus the causes of controversy are demonstrated.

In the present work an attempt is made to describe the organisms in hand as completely as possible considering their morphological, physiological and cultural characters, all at the same time. It has been found that a few media serve well to study all the cultural characters and thus the nature of the growth of the fungus on all these media is described in detail. Certain facts about the reaction of these fungi towards their environment have already been stated and more will be said about it during the description of these organisms.

Note:- For the description of certain terms such as physiologic forms, races, biotypes, cultural characters, and physiological characters see the chapter on Variability.

F.28. Fusarium sambucinum. Fuck. f.5. Wr. ?

Perfect stage: Gibberella pullicaris ?

Syn. Fusarium subpallidum. Sherb. Cornell Univ. Agric. Exper. Sta. Mem. 6,1915. p. 230.

Source:- Diseased wheat plants; obtained from the culm as well as the ear. Isolated in pure culture and subsequently obtained as a single-spore culture. The characters detailed below are those of a single-spore culture grown on different media. /

Only one single-spore culture was used throughout the study of these characters.

Cultural characters and Description:-

Aerial mycelium; abundant, .5 to 1 c.m. high, usually white and cottony on all media when the cultures are inoculated from the mycelium from a normal culture; less abundant when the cultures are inoculated from sporodochia.

MALT AGAR: pure white* to creamy white upto six weeks, attaining a sage tint after that period.

OAT & WHEAT MEAL AGARS: pure white to creamy white when the slants are inoculated from submerged mycelium, dirty white to sage tint when the slants are inoculated from sclerotia or sporodochia.

POTATO-DEXTROSE AGAR: pure white to snow white irrespective of the inoculum.

POTATO (RAW) PLUG: pure white to snow white.

@SALTS-DEXTROSE AGAR: white to light creamy white.

RICE GRAINS: pure white to creamy white with a sage tint at places.

WHEAT GRAINS: the same as on rice grains when young, but acquiring a sage tint very soon.

WHEAT STRAW & EARS: pure white to silvery white.

@ For the composition of this medium see page 42.

Medium; (The colour of the submerged mycelium is similar to the medium in all cases).

MALT AGAR: burnt umber to sepia upto one month, and brownish drab to almost reddish black after about three months. /

*Repert. de Coleurs par Soc. Fr. des Chrysanthemistes.

OAT & WHEAT MEAL AGARS: madder brown to burnt umber, raw umber and finally maroon.

POTATO-DEXTROSE AGAR: olive brown; some times turning sepia on age.

POTATO (RAW) PLUG: the same as on potato-dextrose agar.

SALTS-DEXTROSE AGAR: olive brown to madder brown and finally sage tint.

RICE GRAINS: pure white to creamy white and finally sage tint.

WHEAT GRAINS: pure white to snow white, sage tint and finally light snuff brown.

Sporodochia; produced on all media after about six weeks, 1 to 3 m.m. in diameter, continuing growth for about three months; coloured variously, white, putty colour, buff, cinnamon, and various shades of blue and green.

MALT AGAR: commonly few and large when the cultures are inoculated from mycelium or buff coloured sporodochia; in the latter case appearing rather late; abundant when the cultures are inoculated from greenish or blue sporodochia; differently coloured, ranging from fleshy white to rosy white, pale Blush, salmon flesh, pale buff, Apricot, Horizon blue, sky blue, Bremen blue, blue slate, grey green, and finally dark drab green. There is a greater tendency to form greenish or bluish sporodochia when the cultures are grown at or above 24°C.

OAT & WHEAT MEAL AGAR: abundant on cultures inoculated from /

from mycelium on a medium other than oat and wheat agars, less abundant on cultures inoculated from sporodochia or mycelium on oat agar; commonly putty coloured to Chamois, buff, cinnamon, Hazel, dead leaf, or Horizon blue, blue slate, grey green and finally olive green.

POTATO-DEXTROSE AGAR: Commonly produced on the mycelial mat and minute and aggregated, sometimes large and scattered; sky-coloured white to fleshy white, greenish white, Horizon blue, Cobalt blue, Titmouse blue, blue slate and finally dark drab green or putty coloured to buff and Hazel.

POTATO (RAW) PLUG: minute, blue slate to dark drab green and Holly green; rarely large and putty coloured to buff.

SALTS-DEXTROSE AGAR; similar to those on potato-dextrose agar, but commonly few.

RICE GRAINS: commonly small, snow white to putty coloured.

WHEAT GRAINS: always large, produced inside and outside the grains; mostly fleshy white to putty coloured, buff, cinnamon, and dull brick red at low temperatures, while blue slate to dark drab green, olive green and finally Holly green at or above 24°C.

Pionnotes; typically absent.

Chlamydospores; common on all media, especially so on oat agar and potato-dextrose agar, sometimes giving an Indian lake colour to the medium; mycelial forms intercalary and terminal, occuring singly or in chains or clusters, with clear or /

or granular contents and measuring 10-20 X 9-16 μ; conidial forms frequent in old cultures, occupying all the cells except the two end cells and measuring 6·2-7·5 X 7-8·5 μ.

Thick-walled cells are commonly found in the submerged mycelium and partially desicated conidia. These are not true chlamydospores although they might be mistaken for them.

Sclerotia; very common on oat agar, potato-dextrose agar, rice grains, wheaty grains, and wheat meal agar when the slants are inoculated from the submerged mycelium; rare when the cultures are inoculated from sporodochia; typically absent on malt agar; uaually buff to light brown, dead leaf, burnt umber, raw umber, and finally sepia or smalt blue to blue slate, grey green and finally dark drab green. Formation of sclerotia is favoured when the cultures are grown above 22°C. The sclerotia may be found scattered and having a diameter of 1 to 1 m.m. or in clusters and having ~~a~~ ~~a~~ the size of a small pea.

Microconidia; rare, found on almost all media, 1 to 3 septate, oval or cylindrical, straight or curved, sometimes sickle-shaped and indistinguishable from macroconidia, measuring 3-6 X 18-26 μ.

Macroconidia; sickle-shaped, sometimes cylindrical, slightly pedicellate, often abruptly constricted at the apex, broadest at the middle, walls and septa thick, typically 5-septate, 3 to 6-septate quite common, measuring 24-37 X 4-6 μ; produced in the aerial mycelium and sporodochia.

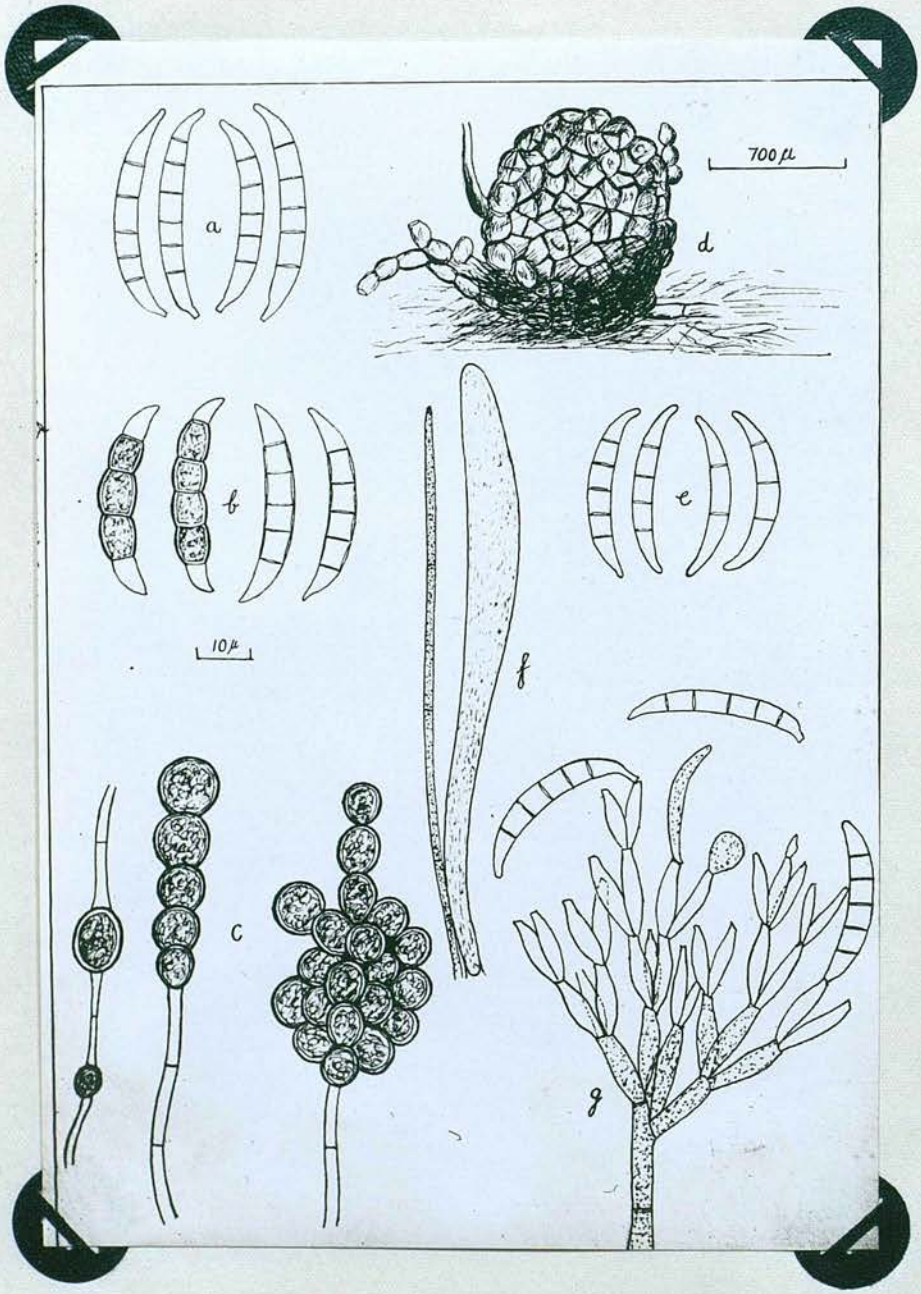

Fig. 9. Fusarium sambucinum. f.5.Wr.

a. conidia. b. conidial chlamydospores.

c.mycelial chlamydospores. d.sclerotium.

e. conidia from malt agar. f. an ascus.

g. conidiophore.

6-septate: rare, sometimes upto 2 %, usually sporodochial; found on all media and measuring 35-37·5 X 5·5-6·1 μ.

5-septate: sporodochial as well as mycelial, sometimes upto 95 %.

On wheat grains; upto 90%, sporodochial measuring 25·5-37·74 X 4·2-6·13 μ.

On oat agar; upto 95%, sporodochial measuring 24-35·5 X 4·5-6 μ.

On potato-dextrose agar; 40 to 60%, sporodochial measuring 25-32·5 X 4·8-6 μ.

Mycelial on all media upto 50 % or even more, and measuring 24-33 X 4·2-5·5 μ.

4-septate: common on all media, and especially in blue or greenish sporodochia on potato-dextrose agar, upto 40 % on this medium; 5 to 30 % on all other media and measuring 24·2-32·2 X 4·2-5·9 μ.

3-septate: common in bluish and greenish sporodochia, less common in buff coloured sporodochia, upto 35 % on potato-dextrose agar and 2 to 30 % on all other media, measuring 24-30 X 4·2-5·9 μ.

2-septate: rare.

1-septate: rare.

0-septate: none observed.

Perithecia; Immature perithecia have been observed on oat agar cultures when they were about three months old. Asci and paraphyses were seen but no ascospores were found. Perithecia, brownish /

brownish, more or less spherical, and measuring 200 to 500μ in diameter; Asci, clavate, curved or straight, measuring 100-125 X 12-15 μ.

All attempts to get mature perithecia met with failure.

This fungus does not grow on basic media and the optimum temperature for its growth lies somewhere near 24°C. However, this fungus can grow fairly well at temperatures ranging between 15 and 27°C. Above and below these temperatures the growth is considerably retarded. Certain facts regarding the influence of the inoculum on the resultant growth have been observed in connection with this fungus and may be stated here one by one;

(a). The production of the aerial mycelium is inhibited when sporodochia or sclerotia are used as inoculum.

(b). The formation of the sporodochia is retarded or inhibited when the inoculum is taken from a culture on a medium similar to that on which the sub-culture is made.

(c). As a rule sub-cultures made from the buff coloured sporodochia do not form sporodochia in the first two generations on any medium, except potato-dextrose agar on which sporodochia, of all the colours mentioned in the description, develop in the first generation.

(d). Sub-cultures from greenish sporodochia develop abundant sporodochia of all colours on all media in the first or second generation.

(e). Sub-cultures from bluish sporodochia behave as normal cultures /

cultures made from the submerged mycelium.

(f). The cultures lose vigour if they are grown on the same medium geheration after generation, while cross culturing on different media keeps up the fungus in normal form.

(g). As a rule sub-cultures made from the aerial mycelium form few or no sporodochia, and sub-cultures made from spores, mycelial or sporodochial, form abundant sporodochia on almost all media.

All the above results have been verified twice and the cultures were made in duplicate.

Some of the above facts also apply to other species of Fusarium with some variations according to the nature of the organism.

The fungus just described does not agree completely with any of the so far described species of Fusarium, but it has a very close resemblence to F.sambucinum. Fuck.f.5.Wr. This latter fungus was first described by Sherbakoff as F. subpallidum. He obtained it from potato tubers affected by rot and also from tubers affected by superficial dry rot. Although there is a close resemblence between these two fungi, they are marked by one very important difference. The fungus described by Sherbakoff does not form sclerotia under any conditions, while my own culture produces these structures in abundance on almost all media. The production of immature perithecia is another difference.The production of blue and green /

green sporodochia is a very unusual character for any member of the section Discolor, and this property is shared by both these organisms.(I have come across only two other species of Fusarium, namely F.graminearum (Gibberella Saubinetii) belonging to the section Discolor and F. lateritium obtained from willow and belonging to the section Lateritium, which form bluish and greenish sporodochia and buff coloured or Apricot sporodochia all in the same culture tube and at the same time.) Since the difference is not very great the present fungus may be placed under the same name pending further confirmation. At present F. subpallidum has been reduced to a form of F.sambucinum by Wollenweber and it is doubtful whether my own organism can be placed under the same form. Since no other form of F.sambucinum resembles the present organism more than form 5, I feel that for the present this organism may be placed under the same form.

Pathogenicity; pathogenic to wheat, barley, oats and rye under the conditions tested. More severe in the Foot Rot phase of the disease than at the seedling stage in seedling blight. The Foot Rot is at its highest when the plants are about two months old.

Optimum temperature for growth; 24°C.

Maximum temperature at which visible growth can take place; 32°C.

Minimum temperature for growth; not studied.

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F. 28 (a). Fusarium herbarum ?

Source:- This fungus arose as a mutant in a culture of F. 28 (F. sambucinum. Fuck. f.5.Wr.) on potato-dextrose agar. This culture was incubated at a temperature of 27°C, but by accident the temperature rose up to 63°C. over-night and so it is not possible to give the exact length of time or the exact temperature at which the culture remained.Some cultures on other media were killed, while others remained alive and when kept at room temperature regained normal growth. In the lower half of the culture on potato-dextrose agar the growth of the fungus appeared somewhat different and when transfered to malt and oat agars a distinctly new fungus with no resemblance to the parent was obtained.This fungus has been kept in culture for five generations and so far it has not reverted back or mutated further and all its characters are fixed at the present moment. The most remarkable feature of this organism is, that it forms abundant aerial mycelium but very few, minute and aggregated sporodochia on potato-dextrose agar, while on oat and malt agars it does not form any aerial mycelium and the production of sporodochia is abundant, so much so, that on oat agar these fruiting bodies appear in pionnotal form. The red colour of the submerged mycelium and the medium in the case of oat agar is another character not shared by the parent. This colouration is absent on all other media. The spores of this organism resemble those of F. herbarum belonging to a different section of /

of Fusarium than the parent. This is a very remarkable example of mutation from one section of Fusarium to another and might be of some help in having a clear conception of the phenomenon of variability and mutation in the genus Fusarium.

The fact that there is no other similar organism in culture in this laboratory disproves any claim as to the origin of this organism as a chance contamination. Single-spore cultures have been obtained from this organism and the characters remain unchanged. Since mutants apparently fixed have been observed to revert back, it is not possible to gaurantee the fixed nature of this organism. As stated this fungus is fixed for the present and does not show any signs of revertion or mutation in any other direction.

Cultural Characters and Description:-

Aerial mycelium; present on potato-dextrose agar only; 2 to 5 m.m. high, thick and cottony, white to creamy white.

Medium;

MALT AGAR: dark fawn to burnt umber; medium retaining its normal colour in the deeper parts.

OAT AGAR: Indian lake to Antique red, Morrocco red and finally Garnet brown.

POTATO-DEXTROSE AGAR: white to creamy white; rarely a little development of Indian lake at the bottom of the tube.

SALTS-DEXTROSE AGAR: similar to that on potato-dextrose agar.

Sporodochia:-

MALT AGAR: abundant, '5 to 1 m.m. in diameter, loosely scattered, buff to dead leaf and finally dull brick red to tan brown.

OAT AGAR: abundant, large, '5 to 2 m.m. in diameter, closely aggregated, often forming pionnotes, cinnamon to dead leaf, dull brick red and finally red ochre.

POTATO-DEXTROSE AGAR: developed on the aerial mycelium; few, extremely minute, closely aggregated, putty coloured to buff.

SALTS-DEXTROSE AGAR: abundant, small, closely aggregated, sometimes forming pseudo-pionnotes, commonly buff to cinnamon.

Pionnotes; typical pionnotes only developed on oat agar near the bottom of the culture slope. Closely aggregated sporodochia often give the appearance of false pionnotes on potato-dextrose agar and salts-dextrose agar.

Sclerotia; absent.

Chlamydospores; none observed.

Microconidia; none observed.

Macroconidia; long and narrow, sickle-shaped, sometimes abruptly bent at the upper end, pedicelate, thin-walled, broadest in the middle and gradually tapering towards the ends; typically 5-septate, 3 or 4 septate quite common.

7,6-septate: rare, sometimes upto 2 % on oat agar, and measuring 67·7-86·65 X 3·4-4·2 μ.

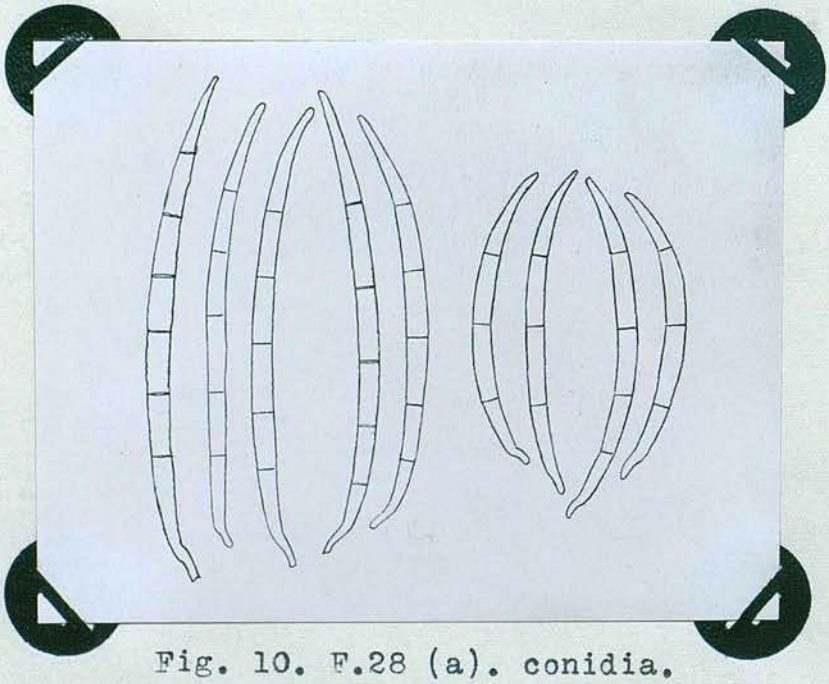

Fig. 10. F.28 (a). conidia.

5-septate: typically sporodochial, 50 to 99 %.

On oat agar; sporodochial or pionnotal upto 99%, and measuring 65·84-86·67 X 2·6-4·2 μ.

On malt agar; sporodochial upto 80 %, measuring 59·31-73·56 X 2·5-4·2 μ.

On potato-dextrose agar; sporodochial and mycelial upto 55 %, measuring 53·2-67·83 X 2·5-4·1 μ.

4-septate: typically sporodochial, upto 50 %, and measuring 51·9-73·4 X 2·6-4·1 μ from oat agar and 40·13-58·2 X 2·6-4 μ. from potato-dextrose agar.

3-septate: typically sporodochial, 5 to 20 %, and measuring 52·6-63·2 X 2·9-3·9 μ from oat agar and 32·6-49 X 2·6-3·72 μ from potato-dextrose agar.

2-septate: sporodochial as well as mycelial, upto 10 % on potato-dextrose agar, and measuring 32·6-40·2 X 2·6-3·8 μ.

1,0-septate: rare.

The characters detailed above show that the organism in hand evidently falls in the species Fusarium herbarum. This is rather a peculiar type of organism and its systematic position should be taken with reserve. I have little doubt as to its place in the species F. herbarum but the varietal place is doubtful. Perhaps it may come under the variety avenaceum.

Pathogenicity; not tested.

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F.29. Fusarium herbarum (Cda)Fr.v.avenaceum(Fr)Wr.

Perfect stage: unknown.

Syn.

(1). Fusisporium avenaceum. Fries, 1823.

(2). Fusarium gaudefroyanum. Sacc.,1880.

(3). Fusarium roseum. Lk.var.lupini albi.Sacc.,1881.

(4). Fusarium avenaceum (Fr) Sacc., 1886.

(5). Fusarium diffusum. Carm., 1893.

(6). Fusarium subulatum. App. et Wr., 1910.

(7). Fusarium pseudoheterosporium. Jacz.

(8). Fusarium lucidum. Sherb., 1915.

Source; wilted oat seedling.

Cultural Characters and Description:-

Aerial mycelium; well developed in normal cultures on all media, 2 to 1 c.m. high; variously coloured.

OAT AGAR: white to rosy pink with patches of Indian lake, dull carmine lake and carmine.

MALT AGAR: rosy pink to bright rose, Indian lake, carmine red, and finally ox's blood red.

POTATO-DEXTROSE AGAR: white to fleshy white.

SALTS-DEXTROSE AGAR: white to lilacy white and Hydrangea pink with patches of Lilac.

RICE GRAINS: white to fleshy white with patches of bright rose, creamy white and Naples yellow.

Medium;

OAT AGAR: carmine red to reddish purple, purple Garnet and finally deep ox's blood red.

MALT AGAR: maroon to dark chocolate brown, brownish red and finally Indian chesnut red or sometimes ox's blood red.

POTATO-DEXTROSE AGAR: normal colour of the medium upto three months or so, and later lilac lake to carmine purple, reddish purple, crimson red, purple Garnet and finally deep ox's blood red.

SALTS-DEXTROSE AGAR: Hydrangea pink to lilac,dark old rose and finally Indian lake.

RICE GRAINS: maize yellow to Naples yellow, straw yellow and finally coppery yellow with patches of rose pink.

Sporodochia; produced on all media, minute or large and upto 6 m.m. in diameter on oat agar, continuing growth for a long time.

OAT AGAR: buff or Apricot to coppery orange, reddish apricot and finally reddish salmon; produced on the mycelial mat and the sides of the tube.

MALT AGAR: buff to apricot and sometimes coppery orange; mostly minute and continuing growth till the medium dries up.

POTATO-DEXTROSE AGAR: pale buff to buff and apricot and sometimes coppery orange; usually minute and aggregated and produced on the mycelial mat especially near the inoculum.

SALTS-DEXTROSE AGAR: pale buff to buff and sometimes apricot; usually minute and aggregated.

RICE GRAINS: none obtained.

Sclerotia; absent.

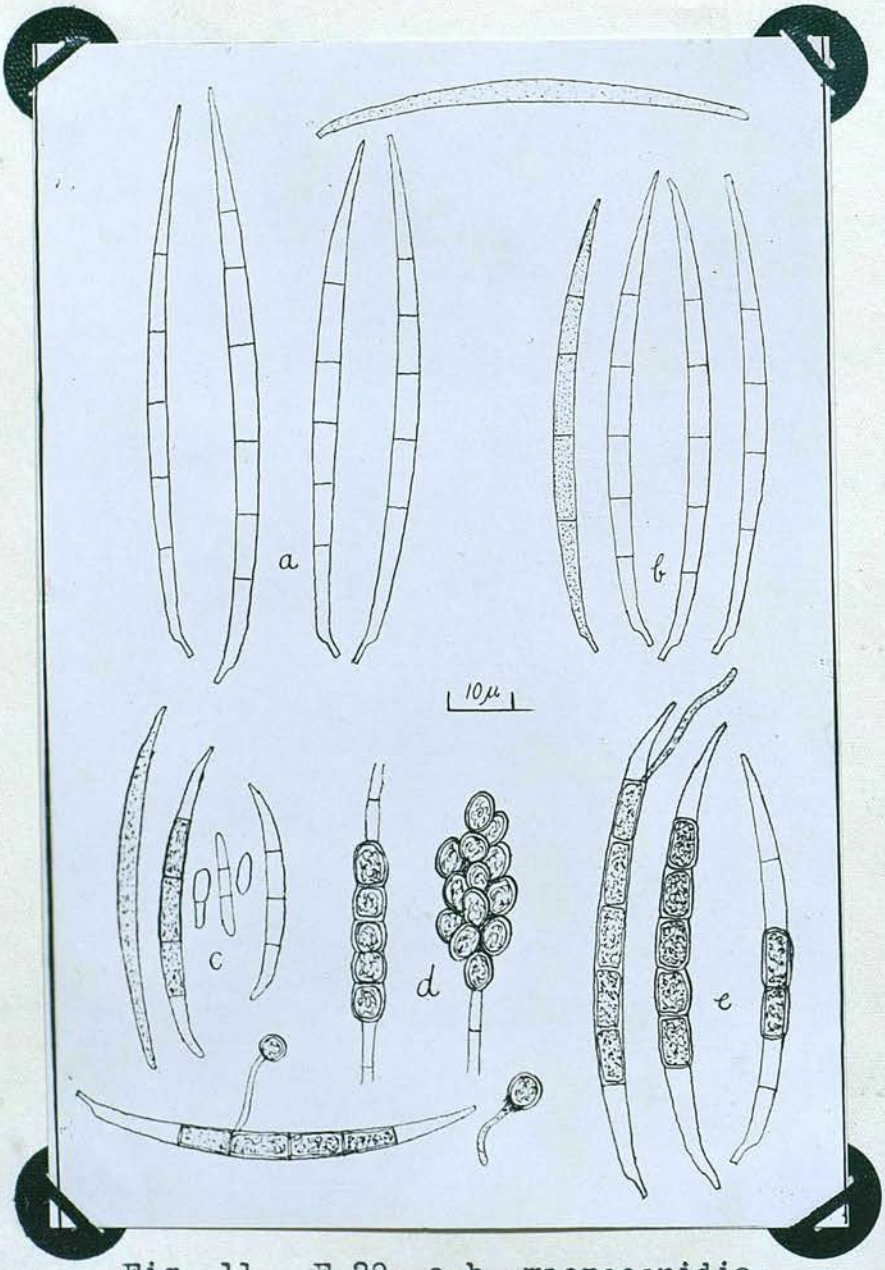

Fig. 11. F.29. a,b. macroconidia. c. microconidia. d. mycelial chlamydospores. e. conidial chlamydospores.

Chlamydospores; present in old cultures, mycelial as well as conidial; mycelial intercalary or terminal, in chains or clusters, measuring 5·3-10·7 X 8·2-13·65 μ;conidial commonly occupying all the cells of the spores except the two end cells and measure 4-4·8 X 7·9-10 μ.

Microconidia; mycelial and sporodochial,1-3 septate, usually of different shapes, sometimes sickle-shaped; present on all media and measure 21·3-50 X 2·5-4 μ.

Macroconidia; mycelial and sporodochial, mostly 5-septate, 3 to 7-septate quite common; commonly sickle-shaped, straight or curved, with parallel walls for the most of the length, prominently pedicellate and having clear or granular contents; highly vacuolate on dextrose media;buff coloured in mass.

6,7-septate: sporodochial; 1 to 5% on oat agar and potato-dextrose agar; absent on malt agar; upto 10 % on plant tissues and measuring 65·48-72·9 X 2·58-4·3 μ.

5-septate: sporodochial as well as mycelial; 50 to 99 %.

On oat agar; sporodochial and mycelial, 70 to 99% and measuring 58·71-72 X 2·58-4·03 μ; average 65·5 X 3·5 μ.

On potato-dextrose agar; sporodochial, mycelial and pseudo-pionnotal, 60-90 % and measuring 52-71·8 X 2·5-4·2μ.

On malt agar; 50 to 80 %; sporodochial measuring 46·3-68·9 X 2·5-4μ.

On oat straw; about 75%; mycelial and sporodochial measuring 57·2-75·5 X 2·5-4·25 μ.

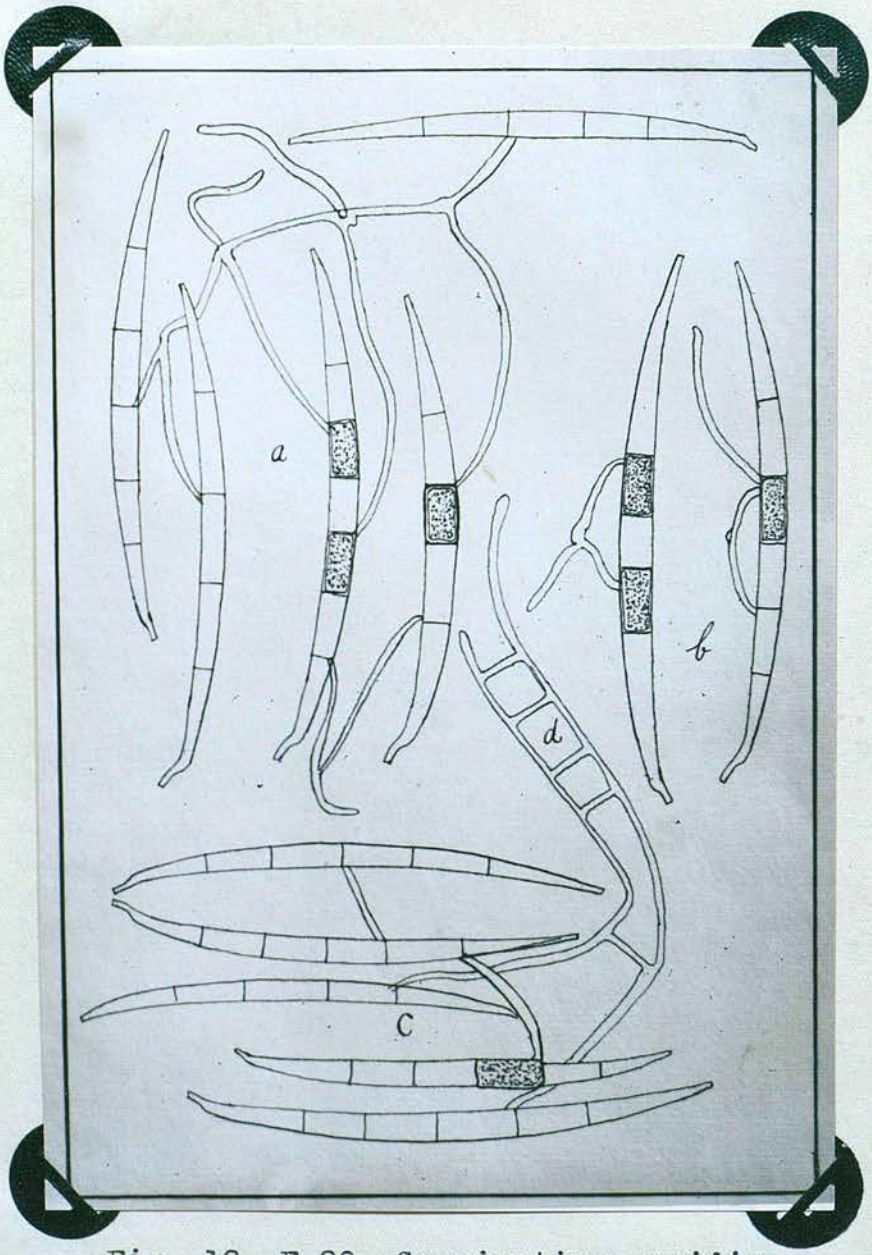

Fig. 12. F.29. Germinating conidia showing various forms of germ tube fusions.

4-septate; 5 to 40 % on all media; sporodochial and mycelial measuring 57·2-67·74 X 2·58-4·03 μ from oat agar; 52-65·8 X 2·58-4 μ from potato-dextrose agar and 40·8-63·2 X 2·5-4 μ from malt agar.

3-septate; 5 to 20 %, found on all media and measure 32·2-50·5 X 2·5-4μ.

2-septate; 1 to 10 %; found on all media, sizes very irregular.

1,0-septate; rare.

This is a very slow growing fungus and is characterised by the early production of colour in the mycelium and the formation of sporodochia within a week. The absence of red colour in the aerial mycelium on potato-dextrose agar is rather unusual. The slight production of a blue slate colour at the bottom of the culture tube in the case of salts-dextrose agar is also curious.

The characters detailed above leave little doubt as to the systematic position of this organism and it can safely be placed under F.herbarum (Cda) Fr. v.avenaceum(Fr.). Wr.

A detailed study of the germination of macro-conidia of this fungus was made and it was found that the germ tubes of many spores fuse together to form a complex thallus from which the resultant mycelium develops. As many as ten spores may be involved in this process of germ tube fusion. Various forms of fusions have been observed and they may be gouped under three heads;

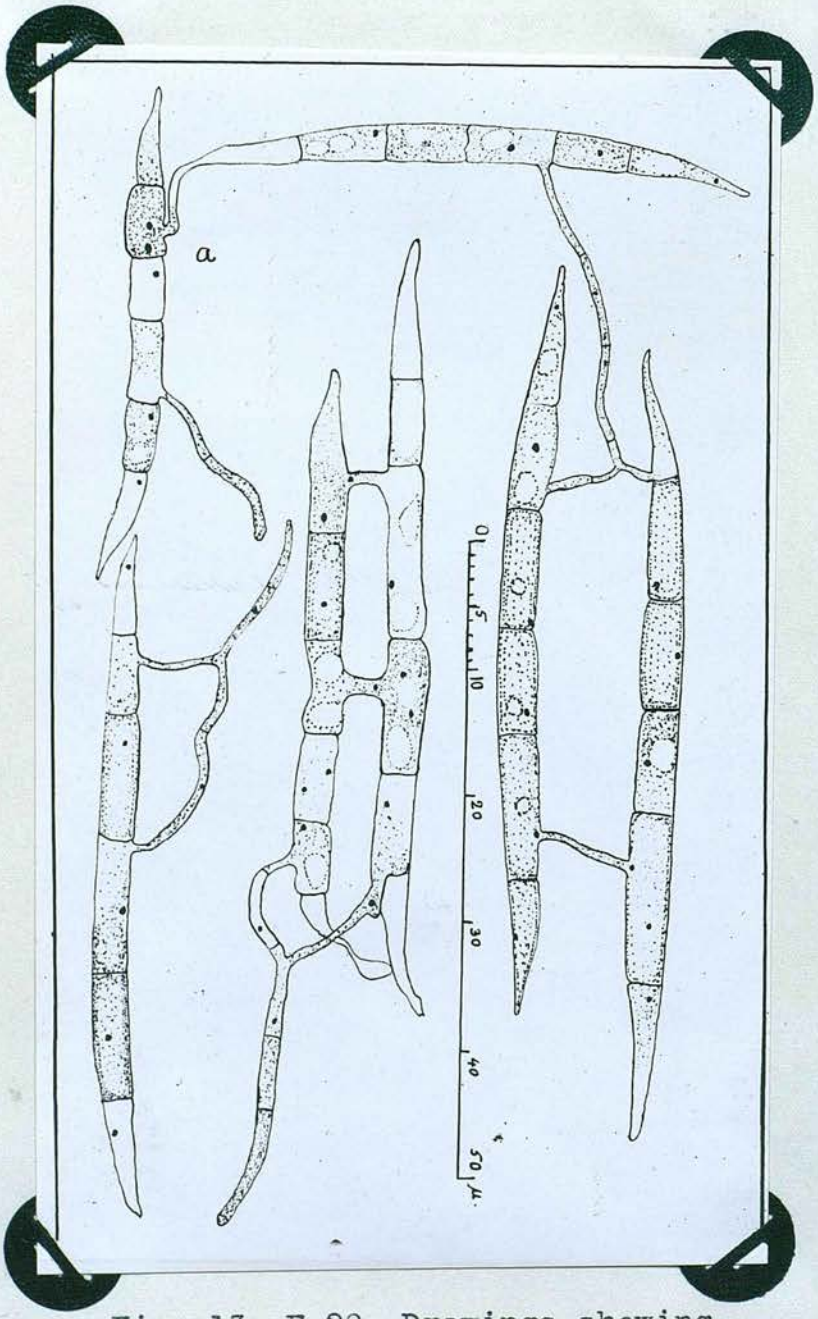

Fig. 13. F.29. Drawings showing germ-tube fusions and transference of the nuclei from one spore to another.

(a). Germ tubes arising from different spores fuse after attaining a certain length and a definite fusion of the protoplasm from different germ tubes occurs. As many as ten germ tubes have been observed to fuse in this manner.

(b). Two germ tubes arising from different cells of the same spore fuse together, and a resultant hypha arises from any of the two cells or from the point of fusion of the two germ tubes. Fig.12.b.

(c). A germ tube arising from one spore fuses with an ungerminated cell of another spore and the contents of the tube pass into this cell, sometimes leaving the germ tube almost empty. There is a definite transference of the nucleus from the germ tube into the other cell, but the two nuclei do not fuse; they just lie side by side as illustrated in Fig.13. a.

The significance of these fusions will be discussed under the discussion of the phenomenon and causes of variability.

Pathogenicity:- Pathogenic to wheat, barley, oats and rye under the conditions tested.

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F.26. Fusarium herbarum. v. avenaceum.

Syn. See F.29. just described.

Source:- Obtained from wheat roots. The crop was suffering from 'foot rot' and 'root rot' and on pulling out the plants, the roots commonly broke off and remained in the soil. Such roots were dug up and the present organism isolated.

Cultural Characters and Description:-

Aerial mycelium; abundant, upto 1.c.m. high, cottony in normal cultures and feathery in non-normal cultures. Shades of yellow absent in normal cultures.

OAT AGAR: white to purplish tinted white or rosy white and cottony in normal cultures; white to creamy white with patches of amber white and lemon yellow, and feathery in non-normal cultures.

MALT AGAR: white to purplish tinted white with patches of Hydrangea pink in normal cultures and white to creamy white and feathery in non-normal cultures.

POTATO-DEXTROSE AGAR: white to fleshy white with patches of pink; usually cottony, sometimes feathery.

SALTS-DEXTROSE AGAR: white to fleshy white; cottony or feathery.

RICE GRAINS: white to creamy white and cottony.

Medium;

OAT AGAR: buff to carmine, crimson red, ox's blood red and finally purple Garnet or creamy white to Naples yellow, amber yellow and /

and finally straw yellow. The red modification is produced in normal cultures and the yellow in non-normal cultures at the same Ph. After about six weeks shades of Antique red and Indian lake appear in the yellow modification and the yellow is totally displaced by these two colours within a fortnight.

MALT AGAR: coral red to Indian lake, crimson red and finally ox's blood red at places or creamy yellow to buff.

POTATO-DEXTROSE AGAR: fleshy white with coral red in patches.

SALTS-DEXTROSE AGAR: creamy white to Indian lake with carmine in patches.

RICE GRAINS: Amber white to cream yellow, straw yellow and finally sulphure yellow at places.

Sporodochia; small and abundant or large and few, produced in all normal cultures on or below the mycelial mat and continuing growth for a long time; sporodochia absent in non-normal cultures.

OAT AGAR: buff or apricot to reddish apricot and sometimes salmon; 2 to 4 m.m. in diameter.

MALT AGAR: buff to reddish salmon and apricot or light carrot red.

POTATO-DEXTROSE AGAR: buff to reddish salmon.

RICE GRAINS: creamy white to amber yellow.

Sclerotia; absent.

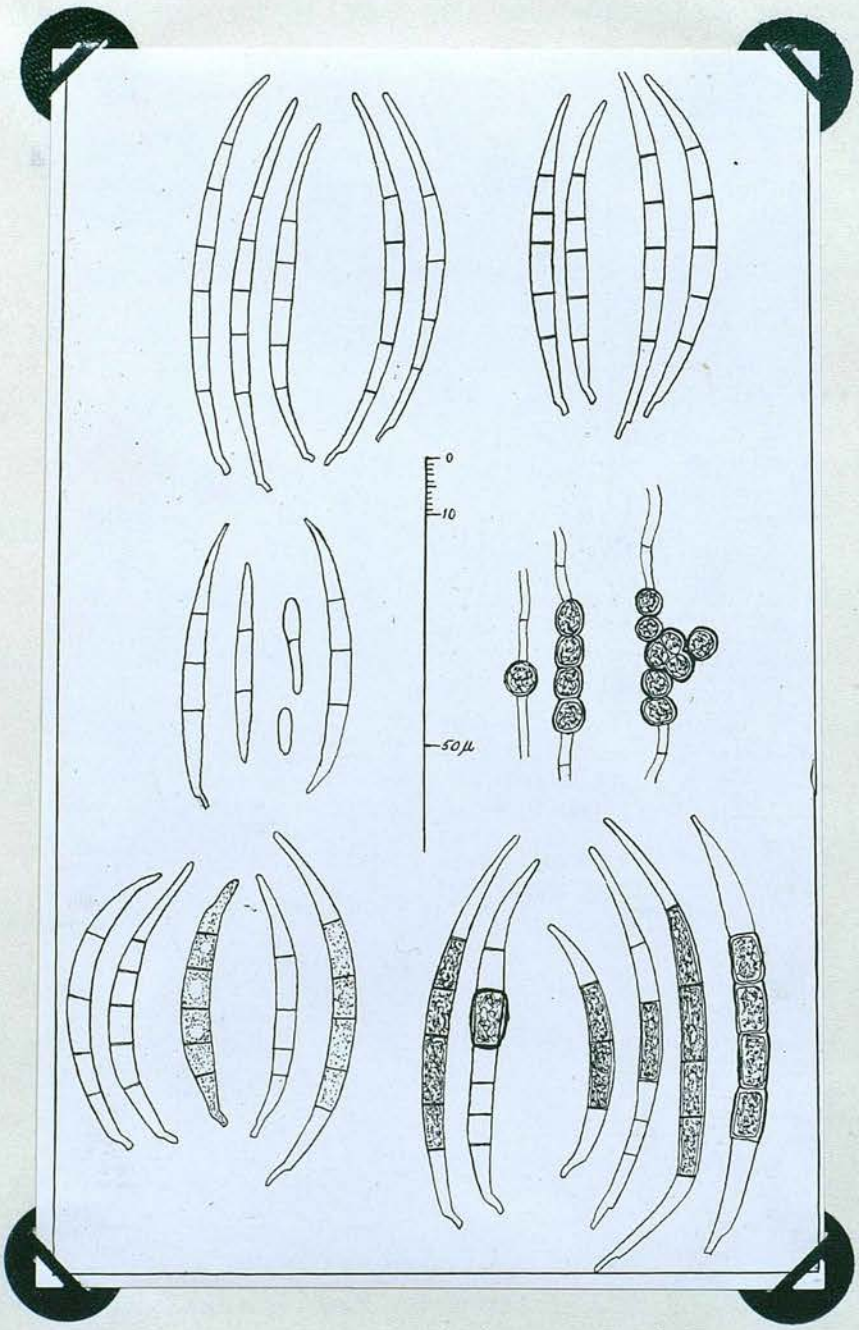

Fig. 14. F.26. Conidia, mycelial and conidial chlamydospores.

Chlamydospores; present in all old cultures, mycelial as well as conidial; mycelial forms mostly intercalary and in chains, measuring 5·2-7·8 X 8·5-10·2 μ; conidial forms ocuupying one, two or even more middle cells of the spore and measuring 3·4-4·9 X 6·3-10·5 μ.

Microconidia; not very common, mostly mycelial, rarely sporodochial and sometimes indistinguishable from macroconidia; septate or non-septate, straight and cylindrical or sickle-shaped and measuring 18-42·5 X 2·5-4·1 μ.

Macroconidia; mycelial and sporodochial, commonly 5-septate, sickle-shaped, pedicellate, broadest in the middle and gradually thinning out towards the ends, usually regularly arched and having clear, granular or vacuolate contents. Average size, 56·8 X 3·8 μ.

6,7-septate: 1 to 3% on oat agar and cereal seedlings; sporodochial measuring 64-67·23 X 2·74-4·51 μ.

5-septate: 50 to 99% on all media, sporodochial as well as mycelial.

On oat agar; 75 to 99%, sporodochial measuring 55·74-66·51 X 2·53-4·51 μ.

On potato-dextrose agar; 50 to 80%, sporodochial measuring 43·2-61·4 X 2·56-4·52 μ.

On malt agar; 50 to 75%, sporodochial measuring 45·4-62·1 X 2·5-4·3 μ.

Mycelial forms 20 to 50 % on all media and measuring 41·3-58·4 X 2·5-4·45 μ.

4-septate: 10 to 50 % on all media, sporodochial/

measuring 38·2-57·87 X 2·34-4·3 μ.

3-septate: 1 to 20 %, sporodochial measuring 27·4-48·77 X 2·4-4 μ.

2-septate: not very common; having the same size as the three septate spores.

1,0-septate: rare.

The characters detailed above show that the fungus is Fusarium herbarum v. avenaceum. The chief difference between this organism and that described immediately before (F.29) is that this fungus grows almost twice as rapidly as F. 29. After the present description of the fungus was written it began to show some signs of variability in the form of sectors. Owing to the lack of time its variability was not studied.

Pathogenicity:- Pathogenic to wheat, barley, oats and rye. The pathogenicity was tested under normal as well as abnormal conditions of growth for the hosts.

The best temperature for the growth of this fungus at which it shows least variation is about 20°C.

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F.1.A,B. Fusarium herbarum (Cda) Fr.

Source:- These two organisms are merely two single-spore cultures derived from a single-spore culture of the fungus F.1.Their cultural characters were studied in detail and it has been demonstrated that even single-spore cultures of the same organism can show appreciable differences when compared under similar conditions, although their source may be the same.It was found advisable to compare the cultures on a large number of media and record the details of differences as accurately as possible.

The fungus F.1. was originally isolated from a diseased sample of oat seed.

Cultural Characters and Description:-

Aerial mycelium;

MALT AGAR: inoculum mycelium; cultures one month old at the time of description.

F.1.A. fluffy and white with pink,rose and straw yellow in patches, and shades of carmine near the medium. Yellow eventually increasing with age. About 1.c.m. high.

F.1.B. white to fleshy white with traces of rose and pink in patches. Mostly flyffy and cottony and about 1.c.m. high.

MALT AGAR:inoculum sporodochia; cultures about one month old.

F.1.A. mostly white with traces of pink and straw yellow in patches. About 5.m.m. high.

F.1.B. creamy white with traces of pink in patches. About 5.m.m. high.

OAT MEAL AGAR: inoculum mycelium, cultures five weeks old.

F.l.A. white to fleshy white and Indian lake with patches of pale gold and flesh coloured yellow.

F.l.B. the same as above.

WHEAT MEAL AGAR: inoculum mycelium.

F.l.A. white to fleshy white and Indian lake with Antique red in patches; compact and cottony and 5.m.m. high.

F.l.B. pink with traces of white and straw yellow; loose and fluffy and about l.c.m. high.

RICE MEAL AGAR: inoculum mycelium.

F.l.A. Indian lake to purple brown with traces of straw yellow.

F.l.B. Antique red to crimson lake with patches of fleshy white and honey yellow.

POTATO AGAR: inoculum mycelium.

F.l.A. white to fleshy white and fluffy.

F.l.B. white to rosy white and cottony.

POTATO-DEXTROSE AGAR: inoculum mycelium.

F.l.A. fleshy white to Indian lake or Antique red.

F.l.B. Hydrangea pink to bronzy old rose with patches of fleshy white and greenish white.

CANE-SUGAR SALTS AGAR*: inoculum mycelium and the cultures about a fortnight old.

F.l.A. white to rosy white, with an occasional patch of rose red and straw yellow; loose and fluffy and about 1·5 c.m. high.

*For the composition of the medium see page 42.

F.1.B. white to purplish tinted white; no shades of yellow. About 1.c.m. high.

LEVULOSE SALTS AGAR: inoculum mycelium.

F.1.A. white to purplish tinted white; 1'5 c.m.high.

F.1.B. white; about 5.m.m high.

LEVULOSE-CANE SUGAR SALTS AGAR: inoculum mycelium.

F.1.A. white to purplish tinted white; 1.c.m.high.

F.1.B. white to lilacy white; 1'5.c.m.high.

LACTOSE SALTS AGAR: inoculum mycelium,& sporodochia.

F.1.A.white to fleshy white, about 5.m.m. high.

F.1.B. white with patches of fleshy white and lilacy white; about 2.m.m. high.

DEXTROSE SALTS AGAR: inoculum mycelium.

F.1.A. white to purplish tinted white; 1.c.m.high.

F.1.B. white with lilacy white at the bottom of the culture tube; about 1'5.c.m. high.

LEVULOSE-DEXTROSE SALTS AGAR: inoculum mycelium.

F.1.A. white to purplish tinted white and Hydrangea pink; about 1.c.m. high.

F.1.B. pure white; about 5.m.m. high.

FRUCTOSE,GLUCOSE,MALTOSE-SALTS AGARS:

F.1.A. white to lilacy white; about 1'5.cm.high.

F.1.B. white with patches of dull rose; about 1.c.m. high.

PEPTONE-SALTS AGAR: inoculum mycelium.

F.1.A. pure white; about 2.m.m. high.

F.1.B. pure white; about 1.m.m. high.

Medium:

MALT AGAR: inoculum mycelium.

F.1.A. Ru ochre to tan brown with patches of Antique red.

F.1.B. Antique red to red salmon and finally blood red.

OAT AGAR: inoculum mycelium.

F.1.A,B. Antique red to dull carmine, morrocco red and finally ox's blood red or Garnet brown.

WHEAT MEAL AGAR: inoculum mycelium.

F.1.A. coppery red to dull carmine lake, crimson and finally Morrocco red.

F.1.B. ochre red to old blood red, carmine lake, and finally ox's blood red.

RICE MEAL AGAR: inoculum mycelium.

F.1.A.,B. dark Indian red to red ochre with patches of carmine and morrocco red. There is a little production of honey yellow in young culturres; later on this is replaced by the reddish colours.

POTATO-DEXTROSE AGAR: inoculum mycelium.

F.1.A. Indian lake to carmine and finally light ox's blood red.

F.1.B. red ochre to carmine lake.

POTATO AGAR; inoculum mycelium.

F.1.A.,B. straw yellow with patches of light Indian lake.

PEPTONE AGAR: inoculum mycelium.

F.1.A.,B. creamy yellow.

CANE-SUGAR, CANE SUGAR-LEVULOSE, DEXTROSE, MALTOSE, AND GLUCOSE SALTS AGARS: inoculum mycelium.

F.l.A.,B. blood red in all cases.

LACTOSE-DEXTROSE AND FRUCTOSE SALTS AGARS:

F.l.A.,B. rosy pink to bronzy old rose and finally Indian lake.

LACTOSE AND LEVULOSE SALTS AGARS: inoculum mycelium.

F.l.A.,B. creamy yellow with patches of bright rose.

Sporodochia: produced on all media in normal cultures, usually large and continuing growth for a long time; buff coloured to various shades of orange.

MALT AGAR:

F.l.A. abundant, 1 to 2 .m.m. in diameter, and reddish salmon in colour.

F.l.B. few, minute and reddish salmon.

OAT AGAR:

F.l.A.,B. abundant, buff to aprict and 1 to 5.m.m. in diameter; produced on the mycelial mat.

WHEAT MEAL AGAR:

F.l.A.,B. abundant, large, yellowish salmon to pale buff, apricot, carrot red and finally capucine lake.

RICE MEAL AGAR:

F.l.A.,B. very few, reddish salmon to Etrusean red; 1 to 2 .m.m. in diameter.

POTATO-DEXTROSE AGAR:

F.l.A.,B. only few, minute, pale buff to dead leaf.

F.l.B. abundant, large, and antique red.

POTATO AGAR:

F.1.A. few, minute, covered with mycelium, buff to salmon.

F.1.B. abundant, large, antique red.

SUGAR MEDIA:

F.1.A. sporodochia of various sizes present on all media except cane-sugar salts agar; commonly buff to apricot, salmon and finally antique red.

F.1.B. comparatively small sporodochia of the same colour as in F.1.A. formed on all media except glucose and cane-sugar salts agars.

PEPTONE SALTS AGAR:

F.1.A.,B. minute yellowish salmon sporodochia develop on drying up cultures.

Pionnotes; typical pionnotes absent.

Sclerotia; minute buff coloured sclerotia were observed on two month old cultures on potato-dextrose agar and wheat meal agar. These bodies are embedded in the mycelium and have a diameter of 100 to 200 μ. When pressed under the coverslip they break irregularly showing white tissue inside the comparatively darker walls. These bodies are not very common and appear to play an insignificant part in the life-history of the fungus. Clusters of chlamydospores form ball-like bodies as described by Bennett for F. harbarum. v. avenaceum, but they can hardly be called sclerotia.

Chlamydospores; common on all media, especially so in oat agar cultures; mycelial forms intercalary and ter-/

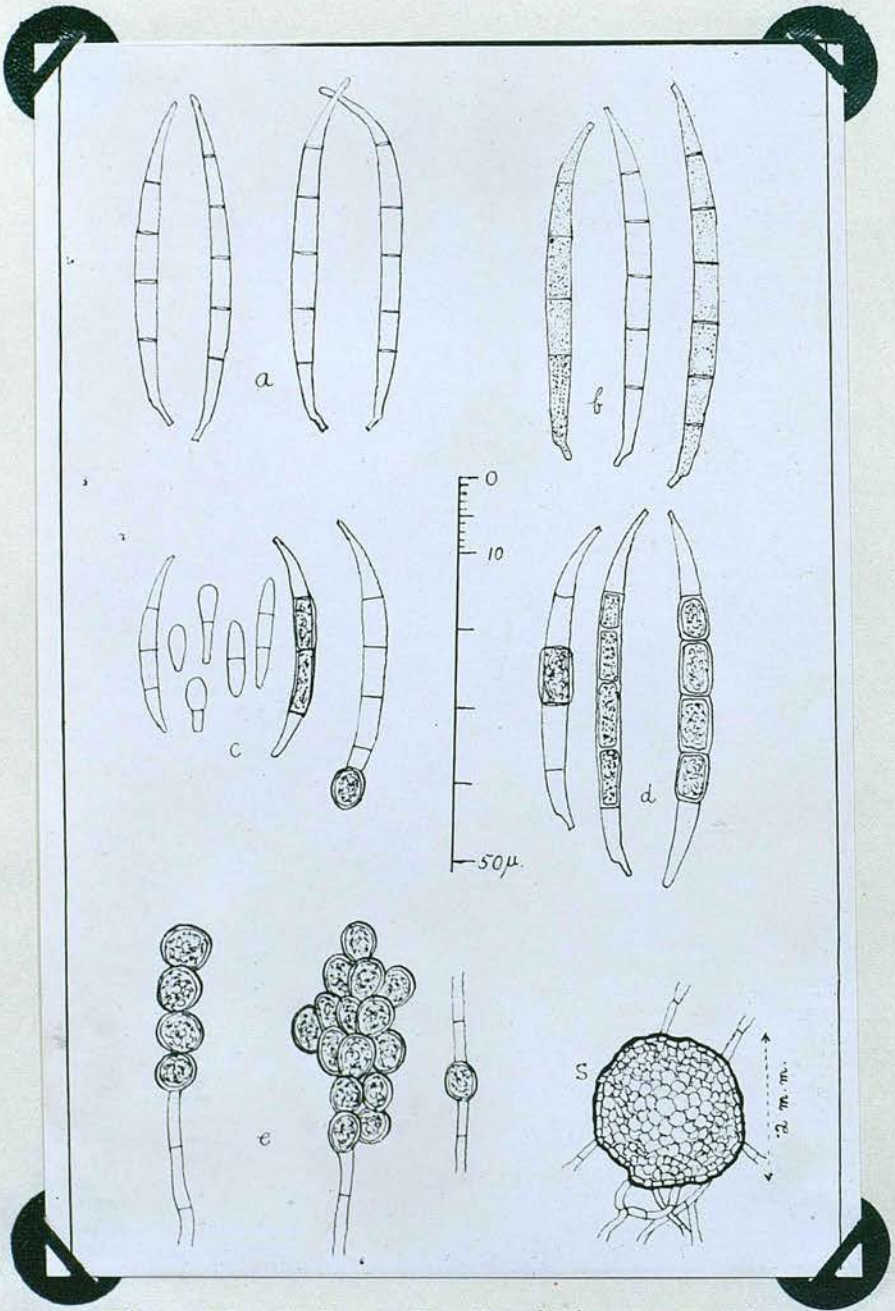

Fig. 15. F.1.A.,B. Conidia, mycelial and conidial chlamydospores, and a sclerotium.

terminal, occuring singly, in chains or in clusters and having a diameter of 7 to 10 μ. Conidial forms occupying two or more cells of the spore measure 4-5 X 8-10 μ. Pseudo-chlamydospores commonly occur in conidia and usually occupy all the cells of the spore except the terminals. These pseudo-chlamydospores are merely comparatively thick-walled cells of the spore with dense and granular contents.

Microconidia: common on all media especially in the aerial mycelium; mostly oval or rod-shaped, sometimes sickle-shaped and 0 to 3-septate.

0-septate: about 10 to 20%, measuring 8-15 X 3-4·5μ.

1-septate: upto 10% and measuring 15-25 X 3-4·5 μ.

2-septate: upto 10% and measuring 20-30 X 3-4·5 μ.

3-septate: resembling macroconidia and measuring 31·5-40·7 X 3·4-3·9 μ.

Macroconidia; mycelial and sporodochial, spindle-shaped or sickle-shaped, upper end abruptly bent in certain cases; thickest in the middle and gradually tapering towards the ends; distinctly pedicellate and showing clear or granular or vacuolate contents; mostly 5-septate.

6-septate: 1 to 5%. most common on plant tissue cultures, less common or even absent on certain agar media such as malt agar. Conidia from oat agar measure 37·7-52·3 X 3·4-4·2 μ.

5-septate: 50 to 99%; sporodochial from oat agar measuring 33·7-50·6 X 3·7-4·1 μ; mycelial from oat agar measuring 31·5-44·2 X 2·9-4 μ; sporodochial from potato-dextrose agar measuring 35·1-46·3 X 3·5-4·1 μ.

4-septate: 20 to 40%; sporodochial from oat agar measuring 35·7-43·3 X 3·4-4·1 μ.

3-septate; 10 to 20%; sporodochial from oat agar measuring 31·6-37·7 X 3·2-4·2μ.

2,1-septate: rare.

The characters detailed above show that the fungus under consideration is Fusarium herbarum (Cda) Fr.

Pathogenicity; a virulent pathogen of wheat and oats; less virulent to barley and rye.

Cultures of this fungus were made on cooked wheat grains and these grains mixed in soil and exposed outside for about sixteen months. The fungus was recovered from these grains after this period. During these sixteen months the fungus had been exposed to two summers and one winter. The summers have been exceptionally dry and the winter also exceptionally cold. In winter these grains on which the fungus was growing had been in contact with snow for about ten days. Under these conditions the fungus did not die out. On examination of the grains large numbers of chlamydospores were found and it appears that the fungus perennates in the form of these spores.

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F.S.2. Fusarium nivale (Fr.) Cesati.

Perfect stage: Calonectria graminicola (B.et B.) Wr.

Syn.;

Calonectria graminicola (Berk et Brm.) Wr.

Lanosa nivalès Fries.

Nectria graminicola Berk. et Brm.

Fusarium minimum Fuckel.

Fusarium nivale autorum pro parte. Scht.

Fusoma triseptatum Sacc.

Fusoma biseptatum Sacc.

Fusarium nivale Sor.

Fusarium hibernans Lind.

Calonectria nivalis Scht.

Fusarium tritici (Liebm.?) Eriks.

Fusarium oxysporum Ces.

Fusarium ustilaginis Rostrup.

Fusarium dimero Fenz.

Source:- Isolated from oat seed, wheat seedling and rye seedling. Many single-spore cultures were made from all the three sources. All the isolations are identical in cultural characters, but the cultures made from the wheat seedling failed to form mature perithecia.

Cultural Characters and Description:-

Aerial mycelium; abundant in normal cultures; absent in non-normal cultures.

MALT AGAR: loose and feathery when grown at or above 17°C.; thick and cottony when grown below 17°C.;white to purplish tinted white, rosy white and finally reddish salmon.

OAT AGAR: abundant, thick and cottony, white to fleshy white, rosy white and finally reddish salmon or salmon.

POTATO-DEXTROSE AGAR: loose and feathery, creeping along the medium; white to fleshy white or rosy white.

SALTS DEXTROSE AGAR: loose and feathery, white to purplish tinted white.

Submerged mycelium and Medium;

MALT AGAR: mycelium white to salmon; medium retaining its normal colour.

POTATO-DEXTROSE AGAR: mycelium white to rosy white; medium retaining its normal colour.

OAT AGAR: white or rosy white to hazel, cinnamon, dead leaf, dark fawn and finally burnt umber.

SALTS DEXTROSE AGAR: white to creamy white, Naples yellow, amber yellow, and finally Gamboge yellow or chrome yellow.

Sporodochia; produced on all media more or less abundantly in all the normal cultures and continuing growth for only a few days. Most common on oat agar, fairly common on malt agar, less common on potato-dextrose and salts-dextrose agar when the cultures are incubated below 17°C. Abundant development of aerial mycelium is essential for the production of plenty sporodochia; sporodochia usually salmon to apricot, reddish apricot, coppery orange and finally dragon's blood. Sporodochia absent in cultures grown at or above 20°C.

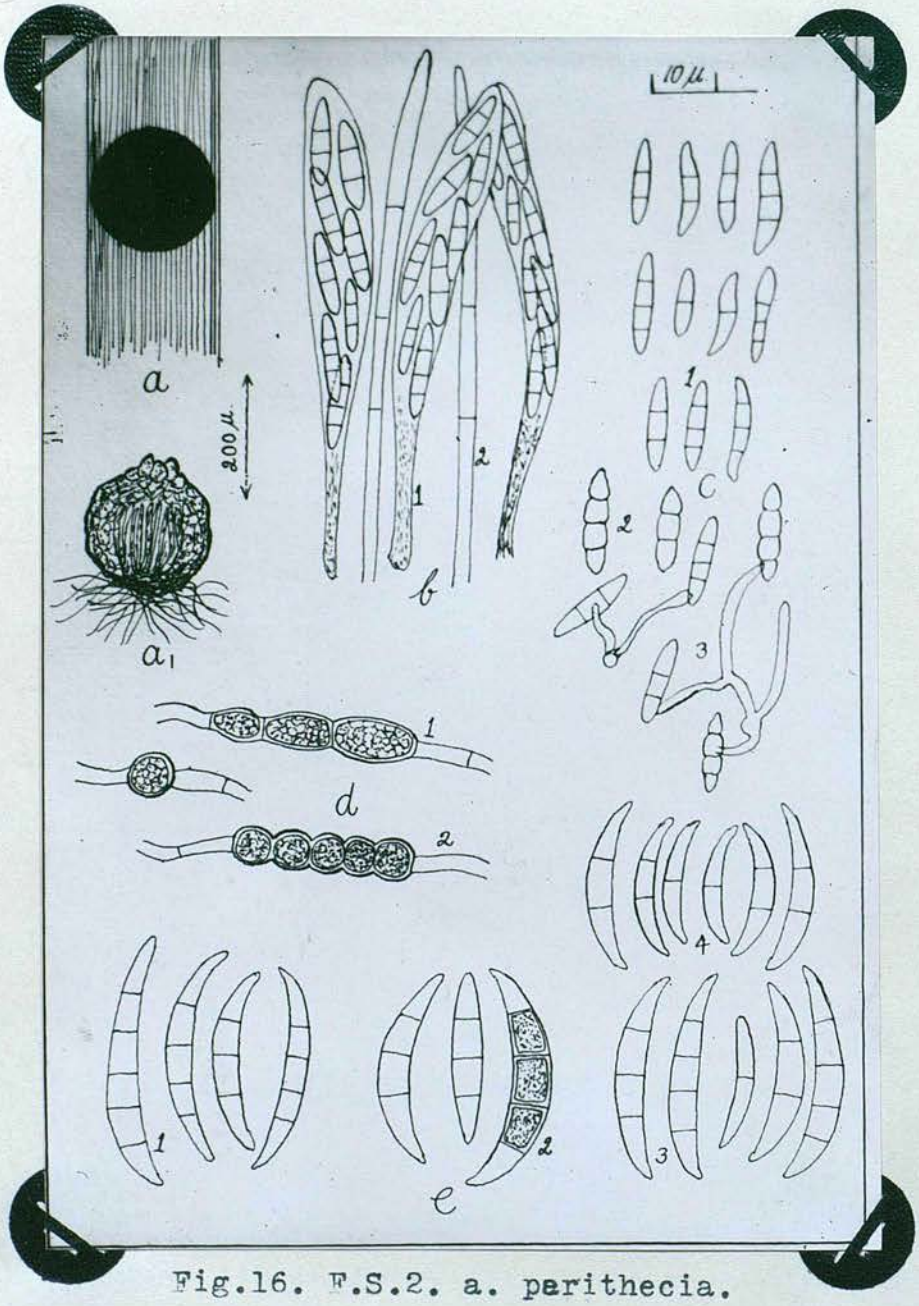

Fig.16. F.S.2. a. perithecia. b. asci. c. ascospores. d.mycelial chlamydospores. e. conidia.

Pionnotes; typical pionnotes not formed in culture on agar media; sometimes the spordochia aggregate to form large compound sporodochia, but these bodies can scarcely be called pionnotes as described by certain authors.Typical pionnotes produced on artificially inoculated seedling (rye) and on soil in the same pot. This pot had been kept in an unheated greenhouse during winter and the fruiting bodies were observed in early March.

Chlamydospores; Typical mycelial chlamydospores have only been observed in one year old cultures on oat agar. These cultures were forming immature perithecia.Chlamydospores absent in cultures forming mature perithecia. Chlamydospores occur in chains and measure 5-7 μ in diameter. Swollen cells of the mycelium should not be confused with typical chlamydospores. Conidial chlamydospores absent. Certain individual thick-walled cells have been observed in very old spores, but these are not true chlamydospores.

Sclerotia; absent.

Macroconidia; sporodochial, comma or sickle-shaped, pedicel absent, 1 to 4-septate, averaging 25 X 3·3 μ.

5,4-septate: not common, measuring 29·3-35 X 3·8-4·1 μ.

3-septate: 30 to 50%; measuring 21-30·5 X 2·8-4·1 μ.

2-septate: 20 to 40%; measuring 20·5-28·7 X 2·5-3·9 μ.

1-septate: 20 to 30%; measuring 18-25·3 X 2·3-3·9 μ.

0-septate: 10 to 20%; measuring 18-24 X 2·3-3·5μ.

Perithecia; commonly free, rarely sunken in the medium, gregarious, light brown to almost black, usually spherical and having a diameter of about 150-285 μ. Perithecia formed only in normal cultures and only about 20% of the perithecia mature.

Asci; cylindrical or club-shaped, straight or curved, having 8 ascospores in two rows and measuring 50-76 X 13-16·3 μ. Paraphyses present.

Ascospores; comma shaped or fusoid, 1 to 3-septate and measuring 12-18·3 X 2-3·3 μ.

The characters detailed above show that the fungus in hand is Calonectria graminicola (B. et B.) Wr. with its conidial stage as Fusarium nivale (Fr.) Ces.

For the production of the perithecial stage it is essential that the culture is brought into the 'norm' characterised by the presence of a well developed, white to salmon coloured aerial mycelium and large number of sporodochia. The perithecia commonly develop on old sporodochia and the sporodochia only develop in normal cultures when they are grown at or below 17°C. The perithecia formed at this temperature do not mature and have to be exposed for a week or so to a cold temperature. Only about 20%. of the perithecia develop ascospores, although as many as 50%.may develop asci. The rest of the perithecia remain sterile under all conditions tested. Mature perithecia have only been obtained on /

on oat agar and rye seedlings. The oat agar cultures were inoculated from normal cultures on malt agar and after incubating all the cultures at 15°C. for a week, some were exposed out-side for a certain period (21-28 November 1932) while the rest were kept at a temperature varying between 15 and 17°C. The exposed cultures produced mature perithecia by 15th of January 1933. The rest of the cultures were also exposed outside after immature perithecia had developed in them. Mature perithecia were obtained after a week's exposure only.

Artificially inoculated rye seedlings bore mature perithecia when they were about one month old. These perithecia were found either on the pionnotes at the base of the seedling or embedded in the leaf tissues.

Cultures kept at or ^above ~~below~~ 20°C. did not produce even immature perithecia, and cultures on raw potato plug, well supplied with water, only formed immature perithecia at room temperature.

The ideal temperature for the production of the 'norm' lies somewhere between 13 and 15°C. The maximum temperature for the development of the vegetative mycelium lies between 28 and 30°C. The optimum temperature for the maximum growth of the vegetative mycelium lies between 24 and 25°C. Cultures grown at these high temperatures atonce change into the abnormal form and must be brought into the 'norm' before sporodochia or perithecia can be obtained. The best method to bring a non-normal culture into the 'norm' is to grow it for /

for one or two generations on oat agar at a temperature of 15°C.

Pathogenicity; All the three strains (isolates) of the fungus are virulent parasites of wheat and rye. Oats and barley are not so badly affected although infection of these two latter cereals may take place upto 90 %. The fungus is more virulent at low temperatures (5 to 15°C.) although it can infect almost any of the above cereals at a temperature of 20 to 25°C.

The temperature best suited to the vegetative growth of the fungus is not really ideal for the infection of the host plant. Successful infections causing serious damage can only occur during cold weather (November to March), while in summer the damage done by this fungus is comparatively less and in certain cases almost negligible.

During the infection experiments with these fungi it was noticed that upto 99 % seedling blight ~~can be~~ was caused by any of these isolates when the infection was carried out in February 1933. On the other hand only 5 to 10 % seedling blight was obtained with the same organisms when the infection experiments were carried out in April 1933. The rest of the conditions were similar. The pots were of the same size in both cases and the number of seeds sown and the amount of inoculum applied was also similar.

So the statements that Calonectria graminicola is a low temperature organism are correct and any contradictions of the above fact are baseless and unjustified.

F.27, F.30. Fusarium culmorum(W.G.Sm.) Sacc.

Syn.

Fusisporium culmorum W.G.Sm.

Fusarium schribouxi Delacr.

Fusarium graminearum Schw.

Fusarium avenaceum Fries.

Fusarium mucronatum Fautr.

Fusarium culmorum MacAlp.

Fusarium corallinum Mattirolo.

Fusarium versicolor Sacc.

Fusarium rubiginosum App.et Wr.

Fusarium heidelbergense Sacc.

Source:- F.27 obtained from a diseased seedling of Victory oats. F.30 obtained from a seed sample of Victor wheat. The two cultures are identical in all respects and thus a single description is given.

Cultural Characters and Description:-

Aerial mycelium; abundant, thick and cottony,upto 1.c.m. high when the cultures are inoculated from submerged mycelium, less abundant when inoculated from sporodochia.

OAT AGAR: white with patches of straw yellow when young, and old rose or rosy pink with patches of dirty yellow Indian lake, antique red, carmine red and ox's blood red when old; the colours eventually deepening down to deep ox's blood red with age.

MALT AGAR: white to violet old rose with patches or scatterings of /

of honey yellow, straw yellow, Indian lake or dull carmine lake and crimson red. Some times these colours are thorough--ly intermixed in minute patches and the resultant colour is raspberry red or Indian lake.

POTATO-DEXTROSE AGAR: hydrangea pink with patches of rosy pink, white and Indian lake.

SALTS-DEXTROSE AGAR: hydrangea pink with patches of white and old rose or light rose.

WHEAT GRAINS: white to rosy pink, Indian lake, and sometimes carmine or carmine red. Shades of dull purple lake, dull brick red and dark Indian red are also developed if the cultures are kept in dark and abundantly supplied with moisture. Cultures kept in sunlight with an abundant supply of water commonly remain white to fleshy white; the red shades vanishing within a week.

Medium:

OAT AGAR: rust red to Turkey red, carmine, crimson red, light ox's blood red and finally morrocco red to purple Garnet or Garnet brown.

MALT AGAR: tomato red to geranium lake, carmine, and finally crimson red. Deeper parts retain the normal colour of the medium.

SALTS-DEXTROSE AGAR: pale rosy pink to bright rose, deep cerise and finally carmine lake; deeper parts retaining the colour of the medium. Near and below the in-oculum the medium is purple black or old olive green.

POTATO-DEXTROSE AGAR: deep cerise to carmine purple, carmine /

carmine, and finally blood red. Deeper parts retain the colour of the medium.

WHEAT GRAINS: grain coats turn dull brick red to brown lake and blood red brown or Indian lake to morrocco red and crimson red. Some grains become almost slate violet.

Sporodochia: usually small, sometimes large, formed on the mycelial mat or on the sides of the medium in drying up cultures and continuing growth for a long time.

OAT AGAR: abundant, large, upto 3.m.m. in diameter; maize yellow to yellowish salmon, apricot, coppery orange, salmon and dead leaf or dull brick red.

MALT AGAR: few and large or numerous and minute; Mars yellow to apricot or buff, dead leaf, dark Indian red, and finally madder brown.

POTATO-DEXTROSE AGAR: abundant, commonly small and produced on the mycelial mat; hazel to buff, apricot, rust red and finally dead leaf.

SALTS-DEXTROSE AGAR: minute, produced on the mycelial mat; hazel to buff and apricot, rarely tan brown.

WHEAT GRAINS: large, produced on and inside the grains; buff to apricot, dead leaf, and finally red ochre or blood red brown.

Pionnotes: typical pionnotes absent; a few aggregated sporodochia form what is usually known as pseudo-pionnotes having the same colour as the sporodochia on the respective media.

Sclerotia: absent.

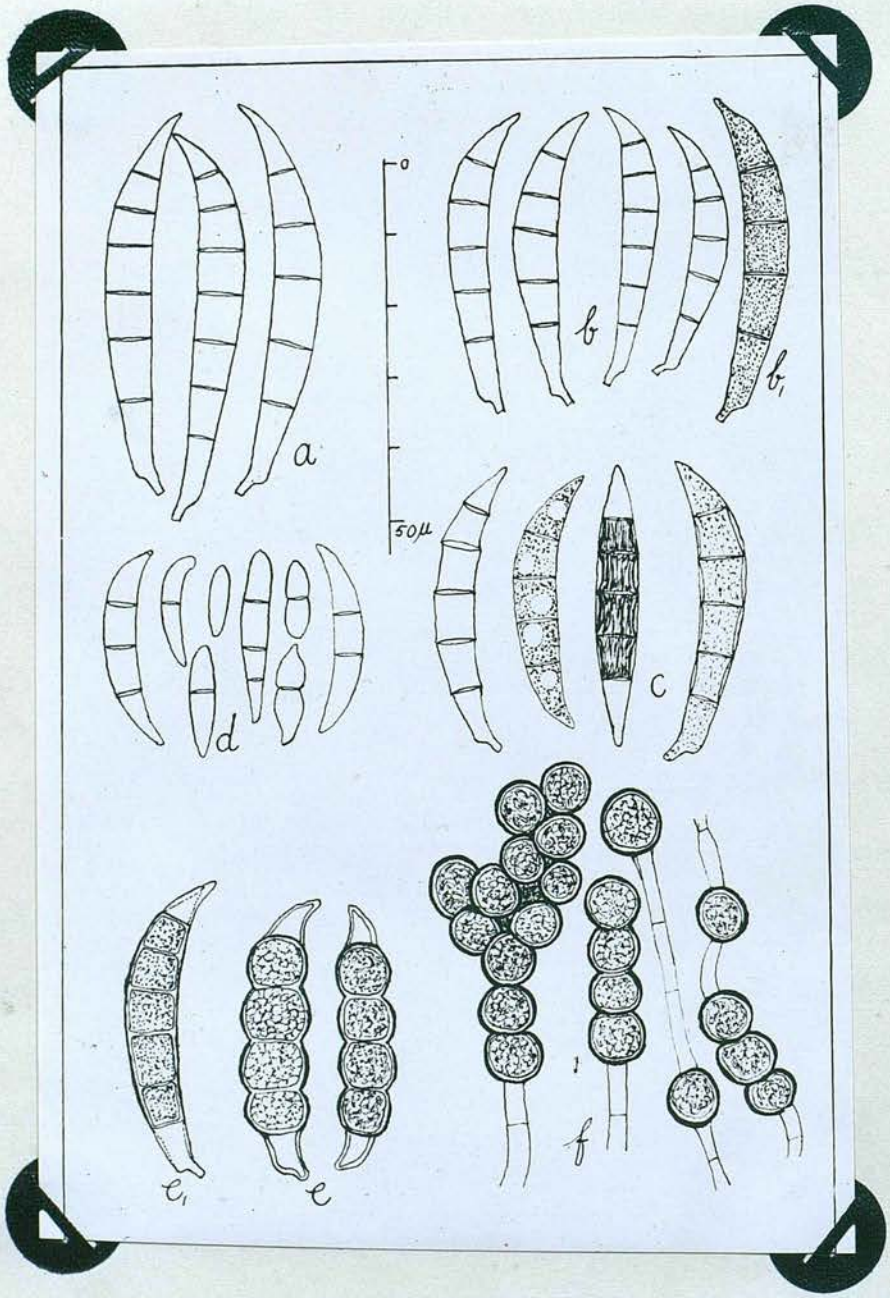

Fig. 17. F.30. a,b,c. macroconidia. d. microconidia. e. conidial chlamydospores. f. mycelial chlamydospores.

Chlamydospores; found on all media in old cultures, mycelial and conidial; mycelial forms intercalary and terminal, occuring singly or in chains or clusters and measuring 6·5-12μ in diameter; conidial forms occupying one or more middle cells of the spore and measuring 8 to 13·5 μ in diameter.

Microconidia; found on all media; commonly mycelial, sometimes sporodochial; usually oval or reniform to spindle-shaped; rarely sickle-shaped and resembling the macroconidia; 0 to 3 septate and measuring 10-24·5 X 3-5μ.

Macroconidia; sporodochial and mycelial, typically 5-septate, 3 to 7-septate quite common; conidia sickle-shaped, prominently pedicellate, comparatively broader in the upper half with apical cell sometimes constricted; conidial walls thick and septa well pronounced; spores commonly colourless with clear, granular or vacoulate contents; some times cinnamon to salmon coloured with granular contents.

7,6-septate: common on oat agar, wheat grains, and wheat straw cultures; sporodochial and mycelial measuring 47·3-54·5 X 6-7·5 μ. (One 7-septate spore measured 63 X7·8μ)

5-septate: upto 80 % on most media, sporodochial and mycelial with an average size of 35·5 X 6·5 μ.

On wheat grains; upto 50 %; sporodochial measuring 32·4-45·7 X 5-7·3 μ.

On oat agar; 45 to 80 %; sporodochial measuring 25·2-45·5 X 5·2-7·1 μ.

On potato-dextrose agar; 10 to 50 %; sporodochial measuring 27·5-45 X 5·2-6·8 μ.

On malt agar; 10 to 40 %; sporodochial measuring 29·3-43·8 X 5·5-7μ.

On salts-dextrose agar; 10 to 50%; sporodochial measuring 30·5-45·4 X 5·5-6·7 μ.

On wheat straw; 20 to 75 %; sporodochial measuring 34·3-48·2 X 5·4-7·1 μ.

4-septate: 10 to 50 % on all media; sporodochial as well as mycelial.

On oat agar; sporodochial 26·3-40·5 X 5·5-6·8 μ.

On wheat grains; sporodochial 27-43·2 X 5·2-7μ.

On potato-dextrose agar; sporodochial measuring 23·5-38·4 X 4·5-6·9 μ.

3-septate: 5 to 20 % on all media, sporodochial and mycelial averaging 24·5-35·4 X 4·5-6·8 μ from all media.

2-septate: not very common.

1,0-septate: rare.

The characters detailed above show that the fungus is Fusarium culmorum as generally recognised in literature. But in view of the facts stated below I very much doubt if this name can be kept permanently. So far as I can ascertain the only difference between the conidial stage of Gibberella Saubinetii (Fusarium graminearum) and F. culmorum is the absence of chlamydospores in the former and the presence of these bodies in the latter. The minor differences in the shape, colour and size /

size of the spores are absolutely false. I have examined two strains of Gibberella Saubinetii for a long time and I have been able to find true chlamydospores in one of these strains. This culture was obtained from the National Collection of Type Cultures and I have no doubt as to the purity and identity of this fungus. I have obtained mature perithecia of G.Saubinetii from the same culture from which the chlamydospores were obtained, and cultures derived from chlamydospores do not differ in any respect from the cultures derived from conidia and ascospores. The culture which formed the chlamydospores was a single-spore culture obtained from the original culture. Every worker on G.Saubinetii has stressed the absence of chlamydospores in this fungus, and many have warned to avoid a probable mistake of recognising certain swollen cells of the mycelium as true chlamydospores. I am fully conscious of this warning and I emphatically state that the structures I have observed are not merely swollen cells of the mycelium but are true chlamydospores. Swollen cells as usually figured, are, of course, quite common in almost any old culture of G.Saubinetii and by comparing these two structures I have been able to avoid any mistake in the recognition of true chlamydospores.Under these conditions I am forced to admit that there is no true morphological difference between Fusarium culmorum and Fusarium graminearum (G.Saubinetii) and that F. culmorum is merely a variety of F. graminearum which has lost its power to produce perithecia of Giberella Saubinetii. This inability to produce perithecia /

perithecia is counter-acted by the capacity of producing chlamydospores abundantly and thus this fungus stands an almost equal chance of over-wintering in the form of chlamydospores as F. graminearum in the form of perithecia (G. Saubinetii).

It must be remembered that any two strains of a Fusarium species can be recognised from their cultural behaviour on different media and thus it is possible to differentiate between F. culmorum and F.graminearum in most cases. Out of the two cultures of F.graminearum (G.Saubinetii) that I have studied one can be picked up and differentiated from F. culmorum even without microscopic examination. This culture forms sporodochia of different colours, namely cinnamon, buff, apricot, and various shades of blue and ~~grey~~ greem. The other culture cannot be easily distinguished macroscopically from F. culmorum in the absence of perithecia, but this strain does not form many sporodochia in culture and no chlamydospores have been found.

It is learnt from literature that all strains of F. graminearum have not the same capacity to form perithecia and some strains do not form these bodies under ordinary conditions. The same is true of F. nivale (Calonectria graminicola). So it is quite possible that the present F. culmorum is a collection of strains of F. graminearum devoid of the capacity to form perithecia.

With these facts in view I suggest that the name Fusarium culmorum be /

be abundoned in favour of F. graminearum, as I have no doubt that these two are merely synonyms of each other. If partition be necessary, F. culmorum may be designated as a variety of F. graminearum which forms chlamydospores abundantly, and does not form perithecia of G. Saubinetii. But it must be borne in mind that the distinction between these two forms will no longer be morphological and qualitative, but merely quantitative in the form of capacity for chlamydospore production.

Since varities of Fusarium species with such differences are known to exist in literature it is advisable to abundon the name F.culmorum and reduce this species to a varietal rank and the name F.graminearum v. culmorum. n.v. be applied.

F. 20. and its saltants.

Fusarium herbarum v. avenaceum.

Source:- Diseased oat seedling.

This fungus has given rise to many distinctly different saltants or variants during the study of its cultural characters and it is doubtful whether the parent itself exists in its true form. All the saltants arose as sectors in plate cultures and so far as possible every effort has been made to isolate them purely and fix them in their respective state. Some saltants reverted back to the parent when grown for a few generations while others remained distinctly different. In this way five distinct forms have been obtained, each differing from the other only in cultural characters and sometimes also in pathogenicity. The morphological characters such as the shape and size of the spores are more or less identical when the cultures are compared on similar media. The capacity to form spores differs in different cases, but all of the saltants form spores either in sporodochia or pionnotes or on the aerial mycelium.

Since the fungus F.20 and all its saltants belong to the species that has been described before in this account of the species of Fusarium its morphological characters need not be repeated. Only the cultural characters are given in detail.

F.20.(parent).

Aerial mycelium: well developed on all media, 2 to 1.c.m. high; white /

white to rosy white with shades of carmine and crimson.

OAT AGAR: white to creamy white, straw yellow or light coppery yellow when young and rosy white to lilacy white with shades of hydrangea pink and Indian lake near the medium after about one month.

MALT AGAR: white to creamy white with patches of straw yellow when young and lilacy white to hydrangea pink with large patches of Indian lake, dull carmine and honey yellow after one month.

POTATO-DEXTROSE AGAR: white to pinkish white.

SALTS DEXTROSE AGAR: white to lilacy white with small patches of sky blue or capriblue near the bottom of the culture tube.

Medium;

OAT AGAR: putty colour to Etrusean red, blood red brown, and finally dark Indian red or Indian lake to dull carmine lake. Deeper parts of the medium retain its normal colour.

MALT AGAR: violet old rose to antique red and finally dull carmine lake or sometimes madder brown.

POTATO-DEXTROSE AGAR: mineral brown to madder brown.

SALTS DEXTROSE AGAR: upper part peach blossom to dark old rose, raspberry red, dull carmine lake and finally crimson red at placed; lower part smalt blue to greyish indigo and finally bluish black.

Sporodochia; pale buff to buff and apricot on all media except salts- /

salts-dextrose agar where smalt blue or greyish indigo sporodochia also develop. Some times sporodochia of mixed colours such as buff and smalt blue (about half and half) are formed in certain cultures.

Pathogenicity; pathogenic to wheat, oats and barley; rye not tested.

F. 121.

Source:- Arose as sector from F.20. in a plate culture on malt agar.

Aerial mycelium; absent on all media.

Medium;

OAT AGAR: carmine to crimson red and finally ox's blood red; deeper parts eventually Vandyck brown to Indian chesnut red and finally mahogany.

MALT AGAR: fawn to madder brown and mahogany; deeper parts deep ox's blood red or purple Garnet.

POTATO-DEXTROSE AGAR: madder brown to dark Indian red or mahogany.

Sporodochia; absent.

Pionnotes; typical pionnotes present on all media.

OAT AGAR: tan brown to rust red, and finally golden reddish brown.

MALT AGAR: buff to apricot, dead leaf and finally madder brown.

POTATO-DEXTROSE AGAR: apricot to red ochre, dead leaf and finally dark reddish brown.

Pathogenicity; non-pathogenic to oats and wheat; barley and rye not tested.

F.123.

Source:- Arose as a sector from F.20.

Aerial mycelium; well developed on all media.

OAT AGAR: white to pinkish white with patches of hydrangea pink and shades of old rose near the medium.

MALT AGAR: white to lilacy white, dark old rose, and finally dull carmine lake.

POTATO-DEXTROSE AGAR: white to pinkish white with patches of old rose.

Medium;

OAT AGAR: carmine to crimson red and finally ox's blood red.

MALT AGAR: carmine to old carmine red and finally deep ox's blood red.

POTATO-DEXTROSE AGAR: carmine to carmine red, old blood red and finally purple Garnet.

Sporodochia; pale buff to buff and rust red on all media.

Pathogenicity; non-pathogenic to oats and wheat; barley and rye not tested.

F.124.

Source; arose as a sector from F. 123.

Aerial mycelium;

OAT AGAR: white to lilacy white with shades of carmine near the medium; '5 to 1 c.m. high.

MALT AGAR: white to pinkish white with patches of pale gold and dark fawn; '5 to 1·5 c.m. high.

POTATO-DEXTROSE AGAR: white to lilacy white, old rose and finally dull carmine lake; 5 m.m. high.

Medium;

OAT AGAR: red ochre to blood red brown, Indian chesnut red, deep ox's blood red, purple Garnet and finally vinous purple.

MALT AGAR: mineral brown to blood red brown, madder brown and finally Indian chesnut brown at places.

POTATO-DEXTROSE AGAR. carmine red to crimson red and finally blood red; shallow parts madder brown.

Sporodochia; pale buff to buff and cinnamon on all media.

Pathogenicity; pathogenic to wheat and oats, barley and rye not tested.

F. 125.

Source; arose as a sector from F.124.

Aerial mycelium; well developed on all media.

OAT AGAR: white to lilacy white with patches of pale gold and Indian lake; pale gold vanishing after about one month. About 1·5 c.m. high.

MALT AGAR: white to hydrangea pink with patches of straw yellow /

or pale gold, light madder brown and buff; 2 to 5.m.m.high.

POTATO-DEXTROSE AGAR: white to lilacy white, old rose and finally dull carmine or crimson red.

Medium;

OAT AGAR: carmine to crimson red, ox's blood red and finally plum violet.

MALT AGAR: red ochre to tan colour, rust red and finally Indian chesnut red or mahogany; shallow parts blood red brown.

POTATO-DEXTROSE AGAR: dull brick red to blood red brown, morrocco red and finally deep ox's blood red or purple Garnet; deeper parts purple brown.

Sporodochia; pale buff to apricot and dead leaf.

Pathogenicity; not determined.

F.127.

Source; arose as a sector from F.20.

Aerial mycelium; well developed, ·5 to 1.c.m.high.

OAT AGAR: white to pinkish white with shades of old rose and dull carmine near the medium.

MALT AGAR: white to lilacy white, old rose and finally Indian lake.

POTATO-DEXTROSE AGAR: white to hydrangea pink.

Medium;

OAT AGAR: carmine to crimson red and finally dull carmine lake or medium ox's blood red.

MALT AGAR: carmine to carmine red, ox's blood red and finally purple Garnet.

POTATO-DEXTROSE AGAR: antique red to ochre red and finally cardinal red with shades of old blood red at places.

Sporodochia; few, large, pale buff to cinnamon.

Pathogenicity; not tested.

F.129.

Source; arose as a fan-like 'sport' from a plate culture of ~~F~~ F.123 on malt agar after the parent had practically stopped to grow further. This 'sport' did not develop any colour either in the aerial or submerged mycelium in the first generation when transfered to different media. It is a very fast growing culture as compared with any other saltant derived from the parent or the parent (F.20) itself.

Aerial mycelium;

OAT AGAR: white to lilacy white with patches of hydrangea pink and Indian lake.

MALT AGAR: white to lilacy white with a shade of solferino red near the medium.

POTATO-DEXTROSE AGAR: white to pinkish white with a shade of hydrangea pink near the medium.

Medium;

OAT AGAR: carmine to dull carmine lake and finally medium ox's blood red; deeper parts blood red brown.

MALT AGAR: fawn to tan colour, dead leaf, red ochre and finally Indian chesnut red.

POTATO-DEXTROSE AGAR: carmine red to crimson red, blood red and /

and finally deep ox's blood red.

Sporodochia; hazel to buff and cinnamon on all media.

Pathogenicity; non-pathogenic to wheat and oats; barley and rye not tested.

Pathogenicity and Infection Experiments.

The pathogenicity of almost all the Fusarium species isolated from cereals was tested and many have been proved to be very serious parasites. There is a remarkable difference in the degree of infection produced by various species under similar conditions, but the pathologic effect is the same in all cases. When I say the pathologic effect is the same I mean that the same phase of the disease produced by different organisms is similar in kind, although there might be a vast difference in the degree to which the phase under consideration is exhibited.

Certain organisms such as F. nivale (F.S.2) and Fusarium herbarum (F.l.A.,B.) are very severe at the seedling stage and during infection experiments it was seen that upto 80 % of the seedlings died as a result of an attack by these two fungi separately. Certain other organisms are more severe to grown up plants and are the cause of a definite 'root rot' and /

and 'foot rot'. F.28.(F.sambucinum) for example, does not produce~~s~~ seedling blight to any appreciable extent, but under the conditions tested it produced a severe'foot rot' when the plants were about two months old.

The following Fusarium species were tested under normal as well as abnormal conditions and were found to be ~~parasitic~~ pathogenic to the cereals mentioned against each;

F.1.A,B. Oats, wheat, barley and rye.

F.26. Oats, wheat, barley and rye.

The following organisms were tested only under normal conditions and were found to be pathogenic to the cereals mentioned against each:-

F.27. Oats, wheat, barley and rye.

F.28. Oats, wheat, barley and rye.

F.29. Oats, wheat, barley and rye.

F.30. Oats, wheat, barley and rye.

F.S.2. Oats, wheat, barley and rye.

F.20. Oats, wheat and barley; rye not tested.

F.124. Oats and wheat; barley, rye not tested.

The following organisms were tested under normal conditions and were found to be non-pathogenic to the cereals mentioned against each:-

F.121. Wheat and oats; barley and rye not tested.

F.123. The same as above.

F.129. The same as above.

The rest of the Fusaria were not tested for pathogenicity.

Since all the infection experiments were similarly carried out, one typical example of each method may be given in order to illustrate the general scheme employed. The discussion of the experiments, as carried out with each fungus, would be merely a repetition of the same account several times.

Infection under abnormal conditions:-

In these experiments the seedlings were grown in Petri dishes and the inoculum employed was a suspension of spores taken from sporodochia. The details of the experiment as carried out with F.26. are as follows:-

Eight Petri dishes with an even layer of sand at their bottom were sterilised in an autoclave at 20 pounds pressure for about half an hour. Then clean samples of wheat (Yoeman), barley (Plumage archer), oats (Marvelous), and rye (Winter) were taken and the seeds surface sterilised by dipping for ~~five~~ 15 minutes in 1 % mercuric chloride and subsequent washing with hot water. Four Petri dishes were sown with 50 such surface-sterilised seeds of each cereal mentioned above and watered with sterile water. These Petri dishes were covered with bell jars and served as controls. The other four Petri dishes were similarly sown with 50 seeds in each, but were watered with a heavy suspension of Conidia obtained from sporodochia produced on oat agar. The spore suspension was obtained by shaking vigorously growing cultures of the fungus containing abundant sporodochia with sterile water and then straining the suspension through a /

a sterilised cheese-cloth. In this way bits of the mycelium were removed and a more or less clear suspension of spores obtained. These Petri dishes were also covered with bell jars and placed along with the rest in the laboratory.

All the rye seeds germinated within four days and the rest of the cereals germinated till the fifth day after sowing. Four wheat seeds (two from each Petri dish sown with wheat), five barley seeds (three from inoculated and two from control Petri dish), and six oat seeds (three from each Petri dish) failed to germinate. The seedlings in all petri dishes were supplied with water containing small amounts of potassium phosphate and sodium nitrate and traces of ferrous sulphate, and in this way reared up for about a fortnight.

During this time only two oat seedlings from the inoculated lot succumbed to the attack of Fusarium and died while the rest carried on fairly well. There was no appreciable difference between the inoculated and uninoculated seedlings except that the inoculated seedlings were a little paler and in many cases the fungus mycelium was growing out of the diseased leaves. No distinct lesions of any kind could be seen on the aerial parts except those places where the mycelium was growing out into the air. These areas were honey yellow in colour but the leaf epidermis was not damaged to form a lesion as is usually formed by Helminthosporium sp.. The bases of the inoculated plants were brownish in colour and in many cases were perfectly rotted. The roots of the /

the inoculated plants were much shorter as compared with the roots of the healthy plants and some of them showed extensive lesions of a dirty brown colour. Certain inoculated seedlings were considerably stunted in the case of oats and a few such are illustrated in plate 12.

At this stage all the seedlings of all the four cereals were cut into small portions of roots and shoots and after external disinfection by dipping in 1 % mercuric chloride for 15 minutes and subsequent washing with sterile water, were incubated in Petri dishes containing malt agar. Majority of the inoculated seedling parts yeilded F.26. and the results of the experiment are summerised in the following table:-

Cereal	Inoculated				Control			
	Number incubated		Number yeilding fungus.		Number incubated		Number yeilding fungus.	
	Shoots	Roots	Shoots	Roots	Shoots	Roots	Soots	Roots.
Wheat	105	92	93	46[a.]	96	85	0+6+1[d]	0+14+0
Oats	106	100	83[c.]	94[b.]	103	104	0+4+8	0+6+5
Barley	93	59	76	44	88	92	0+1+6	0+0+2
Rye	85	76	82	63	69	84	0+5+0	1+7+0

a. 21 root parts gave F.culmorum in addition to these 46 root parts which gave F.26, and 16 roots were run over by bacteria and so the fungus if present did not have any chance of appearing out.

b. The rest of the roots yeilded Helminthosporium.

c. 16 shoots yeilded Helminthosporium sp.

d. In these figures the first number stands for F.26, the second for any other Fusarium species and the third for Helminthosporium species.

The table on the previous page clearly shows that F. 26 can infect all the four cereals successfully under the conditions tested. As is seen in the table, some controls also yeilded various fungi, but this is due to the fact that it is almost impossible to find even a single sample of any cereal in this country that is free from these fungi.During this work I have never come across any seed sample which is free from Fusarium sp. or Helminthosporium sp.

Infection under Normal conditions.

In this set of experiments also, similar methods were employed for testing the pathogenicity of all the Fusaria and thus a typical example will suffice to illustrate these experiments.

Eight six inch pots of sterilised sandy loam soil were taken and sown with wheat (Yoeman), barley (Plumage archer), Oats (Victory), and rye (Winter). Two pots were sown with each cereal and 70 seeds were put in each. Four pots (one pot of each cereal) were left as controls, while the other four were inoculated with F.30. The inoculation was carried out by placing bits of agar with mycelium in the soil at seed level and watering these pots with a light suspension of conidia. All pots were kept in the greenhouse and watered with sterile water as required. In majority of the cases the /

the experiments were carried out in duplicate, and the inoculation of the pots was carried out by adding about 20 gr. of grains, on which the respective fungus had been grown, to each pot. To the control pots was added an equal amount of cooked grains.

All the cereals germinated within a week showing a heavy loss of germination in the inoculated pots. Some of the coleoptiles were badly infected and were brownish in colour. The seedlings were grown for two months and during this time many seedlings wilted from the inoculated lot. Such wilted seedlings died soon after wilting and large amounts of spores were formed on the dead remains. The controls remained fairly healthy and not even a single seedling died during this period.

After this period the plants were cut and small portions of the stem just above the soil level were incubated after extermal disinfection. The roots of the inoculated plants were badly disaesed and large and small lesions of various descriptions were found on about 80 % of the roots. The bases of the inoculated plants were also brownish and when such plants were pulled with a gentle force they commonly broke at the base and the roots did not come along with the plant. The roots and the bases ('foot) of these plants were rotted to various degrees.

All the parts from inoculated seedlings of all the four cereals yeilded the fungus with two exceptions in the case of barley. Only two parts of the control wheat seedlings yeilded the same /

same fungus, and the rest of the cereals did not yeild any fungus resembling F.30., although other species of Fusarium were isolated from oats and barley controls.

The following table gives the results of the experiment in detail:-

The cereal	1x	2x	3x	4x	5x	6x	7x
Wheat,							
Control	68	2	0	0	68	2	3 approx.
Inoculated	59	11	13	5	54	51	95
Oats,							
Control	65	5	0	1	64	0*	1·5
Inoculated	53	17	18·5	8	45	45	100
Barley,							
Control	67	3	0	0	67	0*	0
Inoculated	61	9	9	3	58	49	85
Rye,							
Control	69	1	0	0	69	0	0
Inoculated	58	12	16	6	52	50	96·5

*Other species of Fusarium were isolated from various seedlings.

1x. Number of seeds germinated.

2x. Loss of germination.

3x. Percentage pre-emergence blight due to the attack of the fungus as calculated from columns 1 and 2. e.g. 9 seeds out of 68 did not germinate in the case of wheat due to the attack of the fungus.

4x. Loss of seedlings due to seedling blight.

5x. Number of seedlings (stem parts) incubated.

P.T.O.

6%. Number of seedlings yeilding the fungus.

7%. Approximate percentage of diseased seedlings.

The experiment clearly shows that the fungus can cause a great loss of germination in all the four cereals and that the seedling blight stage may equally be conspicuous in severity.

In this experiment the plants were not grown further to see the effect of the fungus on the maturation of the cereals. Other experiments were, however, carried out to see the effect of Fusarium sp. on cereals as regards maturity. A typical example is cited below.

In this experiment all the four cereals were sown in 12" pots and only ten plants were raised in each. In all 12 pots of sandy loam soil were taken and sterilised in the autoclave. The pots were well watered before sterilisation. Four pots were sown with the four cereals separately and these served as controls. The remaining eight pots were similarly sown with all the four cereals, two pots being sown with each, and inoculated with F.30. The inoculation was carried out by putting about 25 gr. of cooked oat grains on which the fungus had been grown previously. A similar amount of the cooked grains was added to the controls,but no fungus was grown on these previously. The pots were left in the greenhouse and watered as required.

The plants grew fairly well but there was a great difference in the size of the inoculated and the control plants. Rye plants were the first to produce ears and it/

it was seen that the controls which were twice as tall as the inoculated plants, produced perfectly normal ears, while the inoculated plants shot out only a few ears. These latter ears were small and no grain developed in any of the spikelets, but the controls filled normally.

The next cereal to mature was oats. In this case also the controls were about six inches taller than the inoculated plants and produced normal grains in the majority of the spikelets. Some of the control spikelets became bleached but none of them yielded any Fusarium sp.. In the case of the inoculated plants all the spikelets were bleached and only a few formed grain. These grains were perfectly shrivelled and light in weight. All the grains formed by the controls were perfectly normal.

Barley and wheat produced ears about at the same time and results similar to the above were witnessed. The wheat plants that were inoculated produced only three ears and some ears failed to push out of the leaf sheaths. The controls formed normal ears. In the case of barley the inoculated plants did not form any ears, while the controls produced well developed ears.

The foot rot stage of the disease was very prominent in all the four cereals inoculated by Fusarium sp. and a few plants of each cereal actually died before shooting the ears. The bases of such plants were incubated after external disinfection and the fungus was re-isolated from them all. No plants died out of the controls.

In another experiment the inoculation of the ears was carried out with a spore suspension taken from a culture of F.1.A. The ears of wheat and barley and the panicles of oats were sprayed with a suspension of spores obtained by shaking a culture with sterile water and then straining it through cheese-cloth to remove bits of mycelium. The ears were sprayed twice within a week and the controls were sprayed with sterile water at the same time. The ears were left uncovered in both cases and the pots placed in two cornmers separately in the greenhouse. The ears sprayed with the spore suspension became bleached totally or in parts, and the controls remained healthy and green.The detailed description of the symptoms and the course of the disease has already been given else where in this paper and need not be repeated.

The infection of young and mature leaves and nodes of all the four cereals (wheat,barley,oats, and rye.) was also carried out. The leaves and the culms were sprayed with a suspension of spores, while the controls were sprayed with sterile water. The leaf infection was carried out when the plants were fairly small and could be covered with bell jars. After spraying the ~~be~~ plants were covered with bell jars and examined after three days. The places of infection of the leaves were marked by little bleached areas. Such leaves were cut and fixed in acetic alcohol. After all the chlorophyl had been extracted and the leaves properly fixed,the latter were stained with Pianeze III.B. The leaves were not cut into sections /

sections, but were stained as such. The process of dehydration had to be lengthened to suit the occasion and the staining was done in the ordinary way. The hyphae showed up brightly (red) against a green back-ground formed by the stained leaf tissues. In many cases the germ tubes could be traced to stomata, while in other cases they had penetrated the leaf at the junction of two epidermal cells. The course of the hyphae within the leaf tissues was not determined.

In experiments on node infection the nodes were covered with a little cotton wool soon after spraying these parts with spore suspension. The controls were similarly covered after spraying with sterile water. The infection of the nodes took place within three days and they were perfectly rotten within a week. The controls only bleached a little but otherwise remained perfectly healthy.

In all the experiments that were carried out to demonstrate the pathogenicity of the various species of Fusarium, the 'foot rot' and 'root rot' phase of the disease was particularly severe and as a result of the presence of this phase many seedlings died at various stages of their development. The seedling blight phase was also equally prominent. Observations in the fields indicate that the 'deaf ear' and the 'head blight' phase of the disease are also of considerable importance, although much work with these two phases was not done in the laboratory. In a few cases these two latter phases were perhaps the most important of all.

Acremoniella cerealis. n.sp.

This fungus was found twice during the survey of 'Foot Rot' and 'Root Rot' disease of cereals. Last year it was obtained from a badly diseased sample of oats in which the grains were dark, shrivelled and light in weight. The sample showed a very poor percentage of germination. Various seed washings revealed the presence of dark brown, oval or spherical spores which later proved to be those of Acremoniella. Spores of Helminthosporium avenae and some species of Fusarium were also common. The spores of Acremoniella are so similar to the oospores of Phycomycetes like Pythium sp. that at first they were actually mistaken for them. Later studies however revealed them to be absolutely different. Cultures from this source were obtained as described later.

This year this fungus was obtained from a sample of Victor wheat. This was sown in early winter in sterile pots and kept under observation for symptoms of any disease from natural infection of the seed. After about a month a few seedlings wilted and on pulling out of the soil showed typical symptoms of root rot. Microscopic examination of the roots revealed the presence of large numbers of Acremoniella spores on the outside of these roots. In two cases these spores were accompanied by those of Fusarium. The root cortex showed extensive lesions and was almost rotten. Mycelium of Acremoniella was present in all parts of the roots. No mycelium was found in the stem above ground. The dark /

dark colouration of the roots was due to the presence of large amounts of the spores of Acremoniella which when produced on living plant tissues are dark brown. No spores of Acremoniella were observed on the living aerial parts of the host. Isolations from this source were made directly from these spores.

Isolation of the organism.

Isolation from oats:- The grains after external disinfection by dipping in a 2% solution of mercuric chloride for 30 minutes and subsequent washing with sterile water were placed on moist filter papers in sterile Petri dishes and incubated at a temperature of 24°C. After two days a white mycelial growth appeared from about 80 % of the grains from both ends of the seed. Cultures were made from this mycelium on malt agar slopes, and the seeds kept as such for further examination.

Out of the six malt agar cultures so obtained four turned out to be those of Fusarium sp. and the remaining two showed different types of growth. After about a week one of them on examination revealed the presence of minute, cylindrical, uniseptate, hyaline and thin-walled spores. The other culture showed large masses of yellowish brown, oval or spherical, thick-walled spores similar to those observed in the seed washing. From these spores single-spore cultures were obtained by two methods;

(a). Spores from a clean culture were spread on a sterile slide in a sterile drop of water by a platinum loop and the /

the water allowed to dry. Spores that were widely separated from each other were picked up by a fine sterile needle under the low power of the microscope. These spores were either transfered to culture tubes directly or to drops of malt agar on sterile slides. These drops were then examined under the microscope in order to make sure that each drop contained a single spore. Drops with more than one spore were rejected and those containing single-spores were transfered to culture tubes by means of an agar spade. (An agar spade can be made by fixing a piece of broken razor blade in a wooden handle.) In this way very few cultures were contaminated and this method appears to be better than any other method as it avoids any risk of transference of more than one spore to a culture tube.

(b). In this method spores were suspended in sterile water and diluted to such a strength that a single drop taken by a loop of wire showed only four or five clearly isolated spores. From such a suspension a drop was taken and spread on an agar plate. This plate was then examined under the microscope and clearly isolated spores located. Circular discs of agar having single spores were then cut out by a dummy objective which could be swung in place of a real objective after locating the spore, and these discs were re-examined in order to make sure that each contained a single spore. Such discs of agar with single spores were then transfered to culture tubes.

Both these methods are quite useful and clean cultures can be obtained with little or no contamination.

Isolation from wheat:- The methods adopted for isolation were similar to those described for isolation of the fungus from oats except that rough cultures were obtained from the spores found on the roots of the diseased seedlings. From these rough cultures, clean cultures were made by repeated transference of the mycelium or spores and then single-spore cultures obtained from these clean cultures as described for oats.

Cultural Characters & Methods of study.

This fungus can grow on malt agar, oat agar, potato agar and synthetic media and the production of the spores is perfectly normal in all cases. The aerial mycelium is less abundant on oat agar, but spore production is very rapid and large amounts of spores can be obtained within a week. On malt and potato agars the aerial mycelium is well developed and the spore production is also normal. The spores produced on these media are a little lighter in colour than those produced on oat agar or living plant tissues.

For purposes of study single spores were picked up as described under the isolation of the organism, and transfered to drops of malt agar on sterile coverslips. These coverslips were then inverted over sterile glass rings. The edges of the glass rings were smeared with a little vaseline so that the coverslip could be fixed on to the ring. These rings were then placed in Petri dishes with filter papers, having holes /

holes large enough to fit the rings in them, at their bottom. The rings were placed in these holes and sterile water poured in these dishes in order to provide moisture. These Petri dishes were then incubated at a temperature of about 24°C. In this way the spores were kept in close chambers with abundance of moisture. Before incubation every drop was examined to make sure that only one spore was present in each drop. Drops with more than one spore were marked and also incubated in order to see any difference in the resultant growth obtained from single spores and more than one spore in a single drop.

The spores thus incubated at a temperature of about 24°C. germinated after 12 hours giving out hyaline, non-septate germ tubes from the narrower end of the spore. The single germ tube given out by each spore became branched after attaining a length of about 25 μ and soon developed a hyaline mycelium with non-septate hyphae. The thickness of the germ tubes and the hyphae at this age varies between 4 and 5 μ. After five days when the reproductive branches of the fungus are about to come up, the hyphae become septate with vacuolate individual cells measuring 6-7 X 21-60μ. The hyphae anastomose forming H connections and rarely clamp connections. The young hyphae which remain non-septate acquire a bluish tinge at the tips. The hyphae also show marked swellings in places and these swellings are later cut off from the hyphae and the rounded cells thus formed have thick walls. /

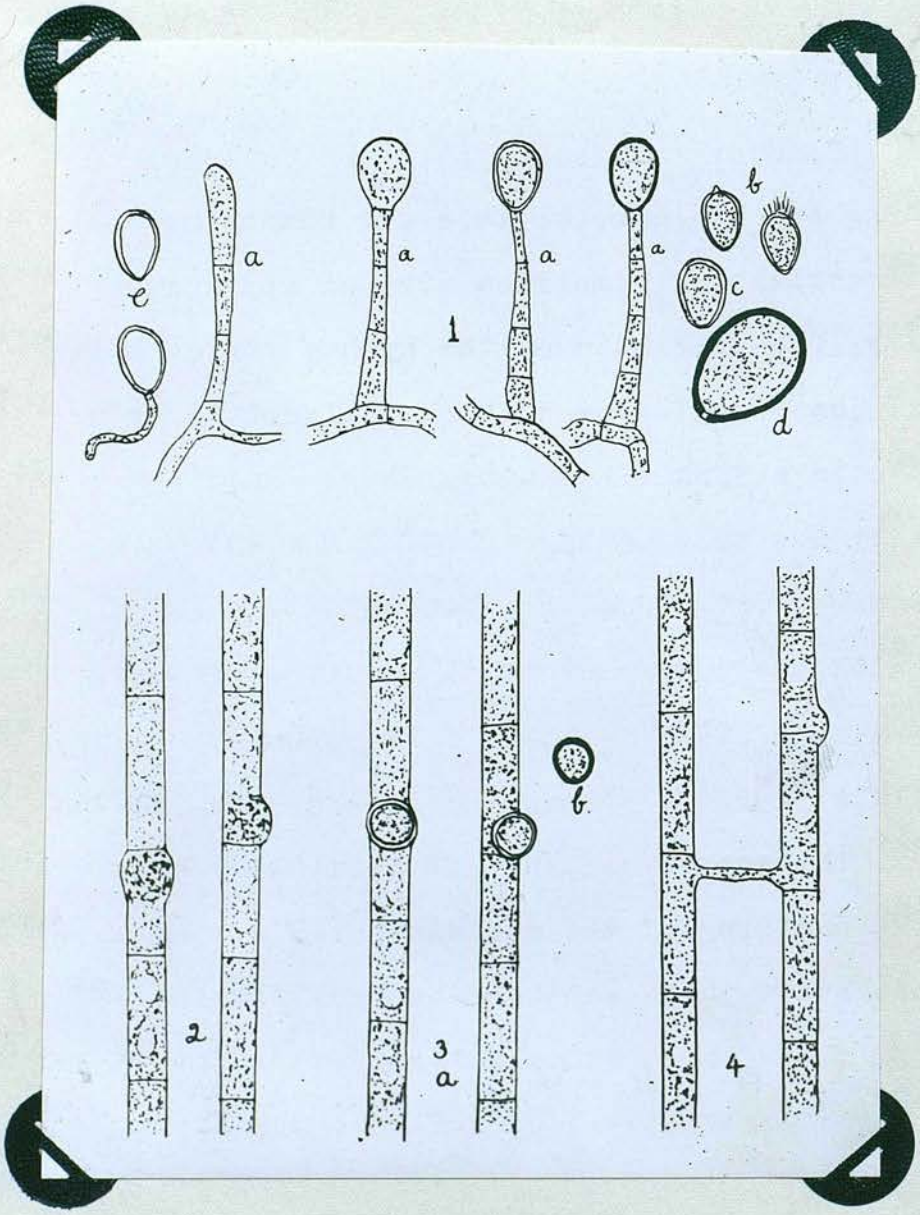

Fig. 18. Acremoniella cerealis.
Conidia, conidiophores, and
hyphae.

The chlamydospores formed in this way have a diameter of 6-8 μ and resemble the round conidia in shape and colour. The tendency to form chlamydospores decreases as the fungus is grown in culture for more than a few generations.

Branches arise on the mycelium after about six days;these are hyaline, septate and branching takes place at acute angles and sometimes give an appearance of false dichotomy. The thickness of the hyphae varies between 5·5 and 7 μ. The conidiophores which are septate or non-septate terminate in a single conidium. These conidiophores are thicker at the base measuring 5·7-7·8 μ and thinner at the top measuring 3·7-4·7 μ. The length of the conidiophores varies with the number of septa and is as follows;

Non-septate 24-36 μ long. Bi-septate 44-72μ long.
Uni-septate 31-34 μ long. Tri-septate 63-72μ long.

The spores are cut off terminally by a septum after the swelling of the conidiophores at the top. This cell develops a thick wall and becomes rounded or oval. When young, the spores have a light yellowish-brown colour and have two distinct walls. Later at maturity the walls fuse and attain a darker shade. At certain stages the spores bear some hairs at the distal end or show a rounded protuberance. These outgrowths which consist only of the thickness of the outer wall vanish at maturity and their significance is not clear. The mature spores are oval or spherical, light brown to dark brown, thick-walled, measuring 21-26 X 17-20 μ, and /

and have a distinct germ-pore at the pointed end. The spores obtained from plant tissues are slightly darker in colour than those produced in culture, but the dimensions do not vary.

The characters detailed above show that the fungus in question belongs to the genus Acremoniella of the family Dematiaceae of the order Moniliales. The genus Acremoniella is placed in the sub-family Monotosporeae of Dematiaceae. The fungus under consideration does not agree with any of the so far described species of Acremoniella. It differs from A. verucosa in having smooth walls at all stages of the life-history, although it has the same dimensions. It has a little resemblance to A.occulta which has been previously described on wheat, but the dimensions of the present fungus are almost double as compared with those of A. occulta. No other species of the organism has any resemblance to the fungus just described. Under the circumstances I feel that a new name should be given to the organism and for that purpose the name Acremoniella cerealis n.sp. is suggested. The host range at present may be limited to wheat and oats only. These hosts are only attacked on the underground parts and thus in this sense also it differs from A.verucosa and A.occulta which attack the aerial parts alone.

Pathogenicity.

In order to determine the pathogenicity of the fungus, infection /

infection experiments were carried out using Sandy and Victory oats and Yoeman wheat as the hosts. Cross inoculations were made with the two strains obtained from oats and wheat. In all 18 pots of sterilised soil were used. The seed was surface sterilised before sowing by dipping in mercuric chloride and subsequent washing with warm water, and 20 seeds were sown in each pot. Two pots of each cereal were sown as such and these served as controls. The remaining one dozen pots were inoculated as follows;

Two pots sown with Sandy oats and inoculated with the fungus obtained from oats. In one pot the fungus was put in the soil and in the other case spores were applied on to the seed.

Two pots sown with sandy oats and inoculated as above with the fungus obtained from wheat.

Similarly the remaining pots were sown with oats (Victory) and Yoeman wheat and cross inoculated as in the case of Sandy oats. All these pots were kept in an unheated greenhouse and watered as required.

The seeds germinated within a week and no signs of disease could be seen at this stage on the coleoptiles. The plants were kept under observation and after about a month some wheat seedlings showed signs of disease. These seedlings began to turn yellowish and within the following week eight wheat seedlings wilted. No seedling wilted in the case of oats. The wilted seedlings from the wheat pots were atonce pulled /

pulled out and on examination revealed large masses of spores of Acremoniella on the roots. The roots were rotten and the fungus mycelium could be seen in all parts of the roots. No mycelium was found in the stem and no spores could be seen on the aerial parts. In the case of two seedlings this fungus was accompanied by Fusarium herbarum and in these cases spores of Acremoniella could be found even on the aerial parts. The fungus was re-isolated from the infected roots when they were incubated after external disinfection as usual. This proved that the mycelium observed in the infected roots was that of Acremoniella and not of any other fungus.

After a fortnight more, some other wheat seedlings wilted and four oat seedlings were also killed. Acremoniella was isolated from all these seedlings, but only from the roots. No fungus could be found in the aerial parts except in the case of oats where Helminthosporium and Fusarium sp. could be found. After this no more seedlings wilted and so the controls as well as the inoculated plants were pulled out and several pieces both from roots and stems incubated after external disinfection. Acremoniella was isolated from a few more roots of wheat and two roots of Victory oats. In the case of oats Acremoniella was invariably accompanied by Fusarium, while in the case of wheat it was found alone as well as in company with Fusarium.

The table on the following page gives the results of the experiment in detail.

	Plants inoculated with the strain from oats.			Plants inoculated with the strain from wheat.		
	Sandy oats.	Victory oats.	Yoeman wheat.	Sandy oats.	Victory oats.	Yoeman wheat.
1*.	20	18	19	20	19	20
2*.	--	--	3	--	--	5
3*.	1	2	4	--	1	3
4*.	--	1	6	1	--	8
5*.	1	3	13	1	1	16
6*.	--	1	13	--	--	14
7*.	1	2	--	1	1	2

1*. Number of seedlings present in each pot during the first week after their emergence from the soil.

2*. Number of seedlings that wilted after a week.

3*. Number of seedlings wilted after a fortnight.

4*. Number of plants yeilding Acremoniella after a month.

5*. Total number of plants infected.

6*. Number of plants infected by Acremoniella alone and only on the roots.

7*. Number of plants infected by Acremoniella in conjunction with species of Fusarium and Helminthosporium.

From the above experiment it appears that Acremoniella ~~x~~ cannot attack healthy oat plants, but wheat can be infected even in full vigour. No difference in pathogenicity could be /

be observed in the two strains of the fungus isolated from wheat and oats. Both strains can attack wheat normally and both can attack weakly oat plants.

Although Acremoniella cerealis can cause true 'Root Rot' its economic importance cannot be very great as it is not very common. The disease is seed borne and the seed is infected by spores that are blown away by the wind from the soil and deposited on the ears. Experiments were carried out for head infection using spores as the inoculum. Wheat ears could be infected upto a fortnight after flowering, while normally healthy oat ears (panicles) could not be infected at any stage of the development. The total absence of the fungus in the stem points out its inability to travel up the stem and reach the ears.

From the above experiments it is concluded that Acremoniella cerealis is a parasite of wheat under normal conditions, and only a weak parasite of oats.

Sclerotial Diseases of Cereals.

Introductory and Historical.

Various fungi with a sclerotium stage in their life-history have been described on cereals and in certain cases they are the cause of 'Foot Rot' and 'Root Rot'. Amongst the chief records of such diseases may be mentioned those of Godfrey from U.S.A., Subramaniam from India, and Samual and Garrett from Australia. The first writer describing a disease of wheat distinguished the causal organism as Sclerotium rolfsii. He reproduced the symptoms of the disease by artificial inoculation from a pure culture of the fungus. His experiments were however crude as the writer himself admits and did not undisputably establish the pathogenicity of the fungus on wheat. But his work is fairly satisfactory to show that Sclerotium rolfsii can infect wheat, and so far as I am aware this is the first record of S.rolfsii on wheat. Later Subramaniam apparently working with the same fungus in India described it under the name of Rhizoctonia destruens, first described by Tassi on Delphinium in Italy. He states that the above two fungi are the same and that the two names are merely synonyms of each other. His statement is confirmed by later workers who compared S.rolfsii and R.destruens under similar conditions and found the two fungi in question to be indistinguishable. Rhizoctonia solani was first reported by Rayllo to infect wheat and oats, but the fungus with which he worked was derived from potatoes and /

and he infected wheat and oats by artificial infection. Peyronel was the first to isolate R. solani from naturally infected wheat. The crop in question suffered from foot rot and the fungus could be isolated from infected roots and culms of wheat. This was his first record in Italy.Recently Samual and Garrett have described R. solani from Australia causing a definite disease of cereal seedlings. They have established the pathogenicity of the fungus on wheat,barley, and oats by infection experiments, and also state that there is no difference between the strains derived from potato and cereals. The disease is stated to be more severe on alkaline "mallee" soils at low temperatures. The disease also affects the ears which remain unfilled and in general the crop is appreciably stunted. Shaw and Ajrekar have reported R. napi. West. on wheat and oats in India. Sclerotium rhizodes has also been described from many countries as the cause of a definite disease of lawn grasses and cereals. This fungus has often been confused with Typhula gramineum by certain European writers, but the two are quite distinct from each other. Braudy's describes the sclerotia of S.rhizodes as subglobose, scarcely glabrous, rough and blackish and those of T.gramineum as irregular, rather flattened and reddish brown. The dimensions of the sclerotia in both cases are similar.

Sclerotium fulvum has been described by Matsumoto on cereals in Japan. He considers this fungus to be identical with T.gramineum and /

and describes the sclerotia of S. fulvum as reddish brown, waxy, spherical or elliptical and ·7 to 2 .m.m. in diameter. They germinate at very low temperatures giving out a white mycelium which can only be grown at or below 0°C. The fungus according to him is unable to grow at 15°C. He was unable to get any basidiospores.

A sclerotial disease of wheat has been described from France by Foex and Rosella quite recently. Their description certainly does not agree with any of the so far described diseases of wheat. They record the presence of violet-amethyst sclerotia in the wheat grains. These sclerotia are covered with a hyphal layer from which endoconidia are given off like ascospores. These sclerotia are white inside and germinate to form a white or deep brown mycelium in cultures. Two types of conidia are produced in culture, ordinary conidia and endo-conidia. The endo-conidia are produced in the interior of the terminal segment of a hypha and measure 6-11 X 3·5 μ.

Various other sclerotial diseases of cereals have been described and in all cases the causal organisms are either placed in the genus Sclerotium or Rhizoctonia. Since these two are merely type genera, there is always a confusion in the taxonomy of such organisms. Then again owing to the inability of certain isolates of these fungi to form the conidial stage or the perfect stage of the fungus new names are invariably given to apparently similar organisms. As a typical example may be cited Sclerotium rolfsii. This fungus has been proved to /

to belong to the genus Corticium and named C.rolfsii. I have studied a strain of this fungus for over six months and I have not been able to get the corticium stage so far. In literature the two names of the fungus are equally persistant. The same is the case with Typhula gramineum. This fungus rarely forms fructifications and the spores have not been obtained by many workers on this fungus. Under these conditions the fungus is confused with Sclerotium fulvum. Some authers are of opinion that the two are identical while others hold them to be absolutely different. Since basidiospores have not been obtained from S. fulvum the point cannot be settled.

In the present investigation five distinct fungi forming sclerotia have been isolated from diseased cereals and in certain cases typical symptoms of 'Foot Rot' and 'Root rot' have been reproduced in the greenhouse by artificial infection with pure cultures. I have no doubt that all these fungi have been noticed by many workers, but I am at a loss to find any satisfactory records of these fungi in Britain. Under these conditions I deem it desirable to record as much information as possible about these fungi.

Sclerotium fulvum Fr.

This fungus was collected in summer 1932 from a wheat field in east Lothian. The crop was badly affected by 'Foot Rot' and 'Root Rot' and most of it was lying on the ground. The disease /

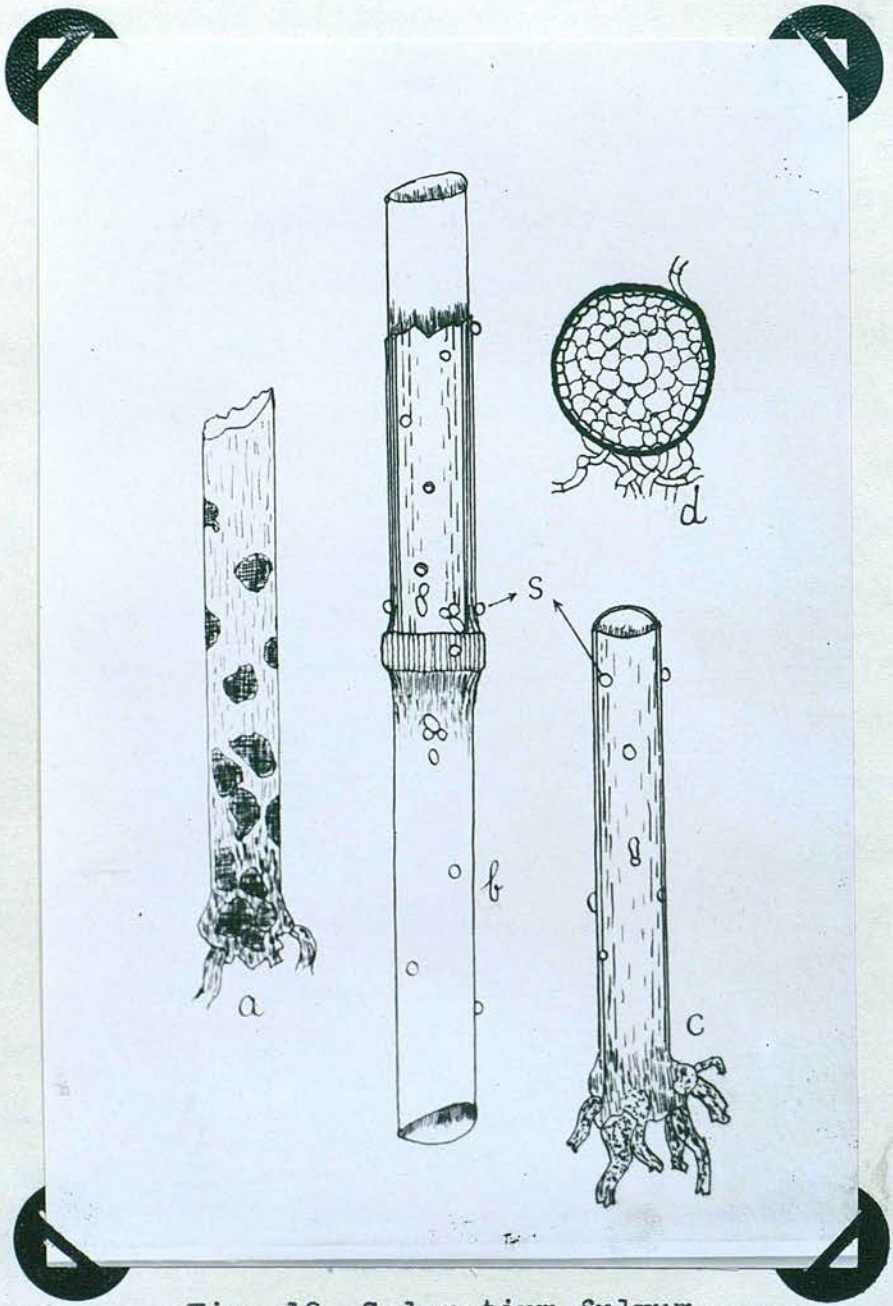

Fig. 19. Sclerotium fulvum.
a. first internode showing discolouration. b,c. culms of wheat showing sclerotia. d. a sclerotium in section.

disease occured in large and small patches throughout the field. The plants were fully grown but bore empty and bleached heads. The normal plants in the same field bore well filled heads which were nearing maturity. The bases of the infected plants were brownish black showing various patterns in the colour shade in different parts of the base. The 'crowns' of these plants were totally rotten and on pulling the plants broke at the crown; the roots invariably remaining in the soil. When dug out the roots showed rotting to various degrees. In less rotted cases distinct dark brown lesions were observed on the root cortex, while in the other cases the rotting had proceeded so much further that the roots were merely a rotten mass of organic matter and crumbled down to pieces when rubbed gently between two fingers. The mycelium of the fungus was found in all parts of the roots, but no sclerotia could be seen.

The discolouration of the aerial parts only extended upto the first node of the stem. A carefull examination of the aerial parts revealed the presence of spherical or oval, reddish brown to orange sclerotia of about the size of a mustard seed. These were only found on the lower nodes, internodes, and leaves, rarely extending upto the fourth node. No sclerotia could be observed on the rotted parts. Feathery tufts of white mycelium were also found in between the leaf sheaths and the stem. This mycelium was organised in poorly defined strands. Various saprophytic fungi and bacteria were /

were also found in association with this fungus and in fact the discolouration of the crown was mainly due to the rot set in by these fungi and bacteria after the death of the host plant caused by the parasite.

Cultures were obtained from Sclerotia after external disinfection by dipping in 1/1 % mercuric chloride and subsequent washing in sterile water. The sclerotia germinate readily when placed on malt agar plated at room temperature and produce a white feathery mycelium which is almost all submerged. A very little aerial mycelium can be obtained by exposing the culture of the organism to a very humid atmosphere. The hyphae remain white on all media upto a fortnight and after this period the mycelium attains a shade of buff to reddish salmon. Sclerotia appear within a week in cultures on malt and oat agars. On malt agar they are first seen as white specks on the surface of the medium in tubes and on sides of the Petri dishes in plate cultures. They develop into white knots of considerable size and take the form of small spheres about .5.m.m. in diameter. Within two days these sclerotia develop a bluish tinge which in certain cases is very conspicuous. They further change colour from bluish white to yellowish white or pinkish white and the later deepens down to orange yellow within two days. This colour continues to be persistant for about a fortnight, after which the sclerotia become dirty brown or mineral brown and remain so till they dry up. On oat agar, however, the sclerotia do not develop as stated on malt agar. The feathery strands /

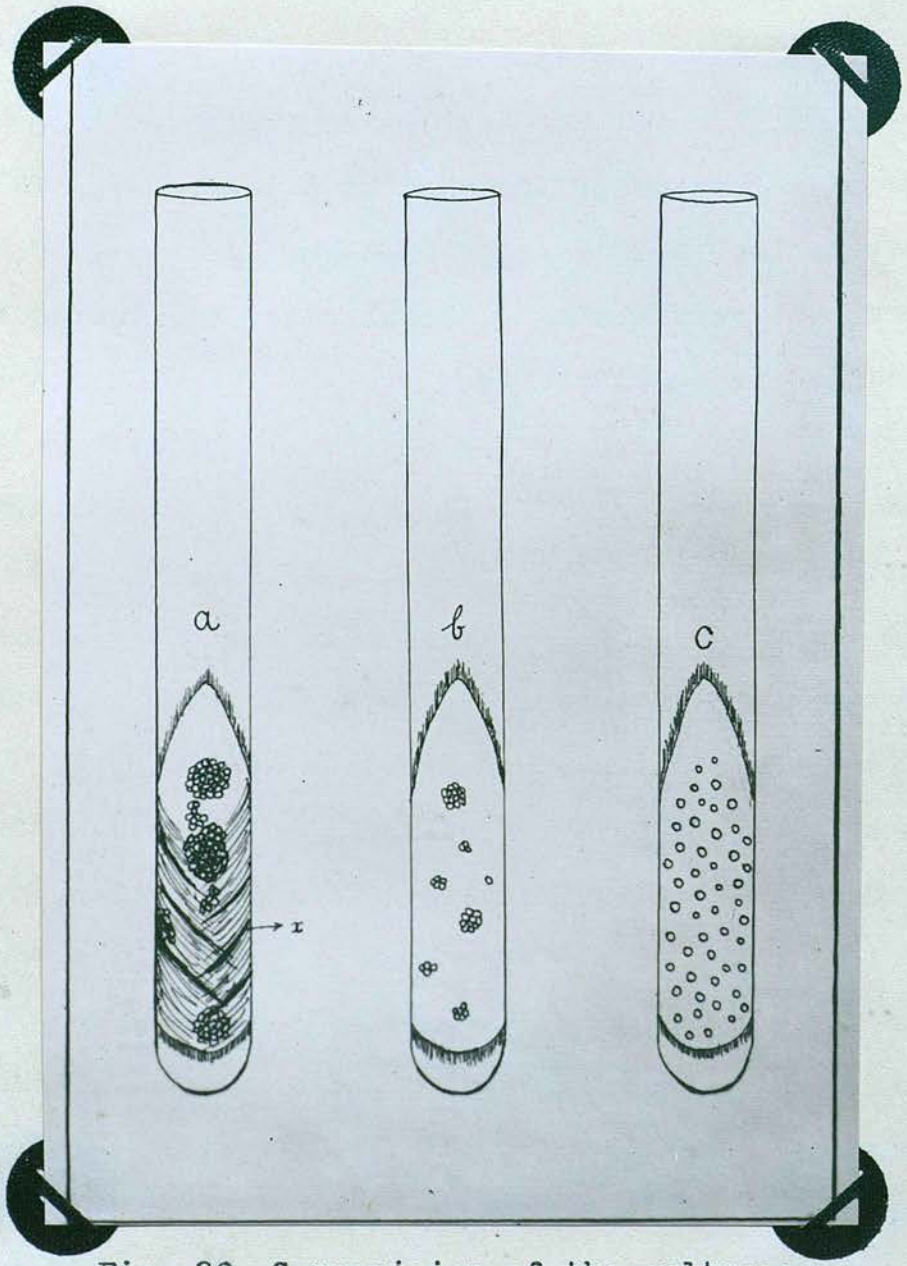

Fig. 20. Comparision of the cultures on oat agar. a. My own culture. b . Sclerotium fulvum. c. S. rolfsii.

strands of the mycelium become honey yellow or buff at places and become inflated by a very rapid division of the cells. This colour deepems to an orange colour and the latter is shortly replaced by orange red. With age the sclerotia begin to turn brown and eventually become dark brown. On both media the sclerotia are aggregated together to form large compound sclerotia of the size of a pea or even larger. On plant tissues they never group themselves into such large masses, but remain single though closely situated. These sclerotia are white inside.

Room temperature appears to be well suited for a good growth of the fungus. The best media for its normal growth are oat agar, potato-dextrose agar, and malt agar. Carrot agar and salts dextrose agar are not very favourable for the growth of this organism.

This culture has been compared with Sclerotium rolfsii obtained from the national collection of type cultures, and S.fulvum obtained from Dr. Butler. These two fungi are absolutely different from the organism in hand and a drawing of the appearance of Sclerotia as produced on oat agar is appended here. The culture of S.fulvum was originally obtained by Matsumoto in Japan from diseased wheat plants. He states that this fungus can only be grown in culture when kept in a refrigerator; it can hardly grow at 15°C, but continues growth at or below 0°C. He was able to obtain white filaments by keeping the cultures in a refrigerator for several months. /

I have grown the same culture at room temperature for over six months and I have been able to get the white filaments from sclerotia formed on oat agar at this temperature. The sclerotia which germinated to form these white filaments were only about one month old and the culture itself was only about four months old. No basidiospores could be obtained.

Though my own culture resembles S.fulvum more than S.rolfsii, it is distinctly different. I have not been able to get any fructifications or filaments from my culture, and the best temperature for its growth lies between 22 and 24°C. Although I have not been able to produce any fructifications from the culture under consideration I beleive it to be a distinct form of Sclerotium fulvum.

Pathogenicity & Infection Experiments.

This fungus is the cause of a serious 'foot rot' and 'root rot' disease of cereals. The plants remain stunted and the ears fail to fill. Apparently the fungus does not produce any pathologic effect on the plants at the seedling stage. If, however, a direct contact is established between the mycelium and the roots, the latter begin to show signs of rot within three days. The aerial parts merely become bleached and do not show any signs of rot.

In order to establish the pathogenicity of the fungus 12 pots of sterilised soil were taken and sown with the cereals wheat, barley, oats and rye. Three pots were sown with each /

each cereal, and four pots, one of each cereal, were left as controls. The remaining eight pots were inoculated by the fungus by placing sclerotia in the soil at different places. The pots were kept in the greenhouse and watered as required.

No signs of any disease could be seen in the inoculated plants upto about two months. After this period the controls kept on making a steady progress while the inoculated plants remained stunted. Within a fortnight after this period typical signs of foot rot were seen in the case of wheat, barley, and oats; rye did not show any signs of disease in this experiment. The fungus was reisolated from all the infected cereals and was found to be identical with the culture used for inoculation. Sclerotia of the fungus only developed on wheat culms and leaves. Only two rye plants yielded the fungus along with Fusarium sp.

In another experiment sclerotia were placed near healthy roots of all the cereals grown in Petri dishes on filter papers. It was found that the young roots became infected within three days and typical dark brown lesions were witnessed within a week. The roots of all the four cereals became infected in this way and the fungus was reisolated from them all after external disinfection. The mycelium penetrates all the tissues of the roots.

The deaf ears stage of the disease could not be very well demonstrated as the pots were too small to carry the plants to the earing stage. However, duplicates from the experiment in which /

which the pots were used for testing the pathogenicity of the fungus,were left as such till the plants came to maturity. During this period many wheat plants died as a result of'Foot Rot' and only a few formed ears. These ears were small and no grain developed in any of them. No oat or barley plant died, but none of them produced the ears.

From the above experiments and observations in the field it is clear that the fungus under consideration can cause a serious 'Foot Rot' and 'Root Rot' disease of the three cereals, wheat, barley and oats, and in certain cases may reduce the yield of the entire crop appreciably.

Rhizoctonia Solani Kuhn.

Rhizoctonia solani was isolated from diseased wheat seedlings affected with foot rot. The crop in general was not very badly infected, although a few patches were extremely yellowish and stunted. Such yellowish plants showed signs of rotting of the bases and when these parts were incubated after external disinfection, cultures of R. solani were obtained in almost pure form. The organism thus isolated did not resemble the common R. solani obtained from potatoes and it was beleived to be a different fungus. Later, however, typical sclerotia were obtained in culture and on comparision with the cultures obtained from potato, the two fungi were proved to be identical.

The strain obtained from wheat forms few but large sclerotia and the colour of the mycelium is a little lighter than that of the potato strain. The wheat strain has a white to dirty white and putty coloured mycelium while the potato strain has a putty coloured to dark fawn or mineral brown mycelium. Both form sclerotia which are mineral brown to raw umber or sepia. The sclerotia are commonly flattened and have a diameter of about 3 m.m., and in certain cases they aggregate to form large ball-like masses with a diameter of 5 to 1.c.m.

The hyphae of the mycelium are extremely vacuolate and those found in the tissues of the plant are colourless. When plated in sterile soil the hyphae become mineral brown and the sclerotia /

sclerotia obtained from this mycelium are purple black. The colour of the hyphae forming the sclerotia is bistre and can be observed under the microscope by pressing a sclerotium under the coverslip. Owing to the lack of time much cultural work could not be done on this fungus.

The fungus can grow on malt, potato-dextrose, and oat agars at a temperature ranging between 15 and 25°C. The formation of sclerotia is most abundant on oat agar and least on malt agar. The sclerotia can resist about six months dessication, when stored in a loosely plugged test tube in the laboratory.

Pathogenicity & Infection Experiment.

Both the strains of R. solani, one obtained from wheat and the other from potato, were tested for their pathogenicity towards cereals. Nine pots of sterilised soil were sown with wheat (Yoeman), barley (Plumage archer), and oats (Victory). Three pots were sown with each cereal and thirty seeds were planted in each. One pot of each cereal was left as control, and one pot of each cereal inoculated with both the strains of the fungus separately. The inoculation was carried out by placing sclerotia and bits of agar with mycelium in the soil at seed level. The pots were kept in the greenhouse and watered as required.

The seeds germinated within a week showing no loss of germination in the inoculated pots. For a fortnight the seedlings grew well, but after this period the inoculated plants began to /

to show signs of disease. These plants began to turn yellow and a few plants in each pot succumbed to the attack of the fungus and died soon after. Some of the diseased plants were pulled out and the roots on examination showed well developed dark brown lesions on the cortex. Some of the roots broke off at the point of infection and remained in the soil when the plants were pulled. Both strains were almost equally severe and both were re-isolated from the diseased roots of all the three cereals. The controls did not yeild any fungus like R.solani. In the case of infected plants no lesions of any description could be seen on the leaves or the culms. The plants were cut after about five weeks and various parts of the seedlings were incubated after external disinfection. The following table gives the results of the experiment in detail:-

Cereal	1*	2*	3*	4*	5*
Wheat					
Control	29	0	0	0	0 %
Inoculated (Wheat Stn.)	28	2	9	21	82 %
Inoculated(Potato Stn.)	28	3	7	17	71 %
Oats					
Control	30	0	0	0	0 %
Inoculated(Wheat Stn.)	29	4	13	20	83 %
Inoculated(Potato Stn.)	28	3	11	21	86 %
Barley					
Control	30	0	0	0	0 %
Inoculated(Wheat Stn.)	29	1	12	22	79 %
Inoculated(Potato Stn.)	30	3	17	25	93 %

*Please see next page.

1*. Number of seedlings in each pot.

2*. Number of seedlings dead within five weeks.

3*. Number of seedlings apparently diseased.

4*. Number of seedlings yeilding the fungus.

5*. Approximate percentage of diseased seedlings.

The table on the previous page shows that the fungus under consideration is pthogenic to all the three cereals tested and can cause a very high percentage of infection. There is no appreciable difference in the pathogenicity of the two strains tested.

The pathogenicity of the wheat strain towards potatoes was also tested and it was demonstrated that the fungus can infect potato tubers. Five healthy potato tubers were taken and their skin injured at places. Four of them were placed on a culture of the fungus on cooked wheat grains and covered with bell jars. Dishes of water were placed under the bell jars in order to keep the enclosed air moist. The fifth tuber was placed on ordinary cooked wheat grains and similarly covered. This latter tuber served as the control. The tubers were surface sterilised before use by dipping in mercuric chloride solution and subsequent washing with sterile water. The inoculated tubers became infected within ten days, and sections of the infected parts showed that the fungus had penetrated about 1·5 c.m. deep into the tuber. Rhizoctonia solani was isolated from the infected tubers after external disinfection of the parts incubated. The control did not yeild R. solani.

Epicoccum tritici P.Henn.

Three strains of Epicoccum tritici have been isolated from wheat, oats and couch grass. These three strains are distinctly different and the chief difference lies in the production of yellow colour in the mycelium and formation of spores and sclerotia. There does not appear to be a great difference in the pathogenicity of the three strains and all the three strains are weakly pathogenic to the cereals tested. Owing to the fact that the fungus under consideration is not economically important its cultural characters were not studied in detail. However, a little description is given below in order to have a little idea of these fungi.

Cultural Characters:-

Wheat strain:-

Aerial mycelium; white to creamy white or straw yellow; thick and cottony and about 5 m.m. high.

Submerged mycelium & medium; snuff brown to raw umber, deep sepia and finally purple black.

Oat strain:-

Aerial mycelium; white to creamy white and honey yellow; the yellow colour vanishing in old cultures; thick and cottony and about 1.c.m. high.

Submerged mycelium & medium; fawn to tan colour, red chalk, and finally madder brown to mahogany at places.

Couch grass strain:-

Aerial mycelium; white to creamy white, honey /

honey yellow, lemon yellow and finally chrome yellow with patches of coppery yellow and reddish apricot. In certain cases patches of reddish salmon and dragon's blood appear in young cultures.

Submerged mycelium & medium; honey yellow to Indian yellow or chrome yellow, yellow lake, orange cadmium, Japanese yellow and finally red lead in parts; deeper parts morrocco red to maroon and dark chocolate brown.

The above description of the fungi is given from cultures grown on oat agar.

The oat and wheat strains form spores in culture while the couch grass strain has not produced any spores in culture so far. There is no appreciable difference in the size, shape or colour of the spores produced by wheat and oat strains. The spores are produced in dark brown to almost black sporodochia. The spores are light brown to dark brown, oval or spherical, mostly unicellular, often bi- or tri-cellular, with verucose walls and measuring 15 to 25 μ in diameter. Some times a part of the conidiophore remains attached to the conidium. When placed in water the spores germinate within 12 hours giving out hyaline germ tubes from each cell.

The sclerotia commonly develop on all media, but the wheat strain forms very few of these bodies. The sclerotia are dark brown to almost black and measure 100 to 1000μ in diameter. The sclerotia are white inside.

Pathogenicity & Infection Experiments.

All the three strains can infect healthy plants/

plants of wheat, barley and oats. No serious damage, however, appears to be done by the attack of these fungi, and the plants grow as if uninfected. In nature the fungus is usually associated with species of Fusarium, Helminthosporium, and Erysiphe, and can be found in almost any part of the infected plant. It has been isolated from grains of wheat and oats, leaves of wheat, oats and couch grass, and stems of wheat. The fungus was never found in the roots of ~~the~~ naturally infected plants. The characteristic feature of the attack by this fungus is the formation of very conspicuous and well developed dark brown lesions on the infected parts. On grains the point of infection is marked by a discoloured area instead of a lesion. The discolouration of the grains is usually dark brown and they appear as if smoked. The infected grains are capable of germination and the fungus does not interfere with the normal development of the resultant seedling. Well marked lesions may be seen on the coleoptiles, but the first leaf commonly escapes infection.

In order to establish the pathogenicity of the three strains of the fungus similar experiments were carried out with all the strains. The following example illustrates the method of infection employed in these experiments.

Six pots of sterilised soil were taken and three of them sown with wheat, barley and oats separately. These three pots served as controls. The other three pots were similarly planted with the seeds of wheat, barley and oats, but they were /

were inoculated with the wheat strain. The inoculation was carried out by adding about 20 gr. of wheat grains, on which the fungus had been grown previously, to each pot. The pots were kept in the greenhouse and watered as required.

Within a week all the seeds germinated and no signs of any lesions were seen on the inoculated coleoptiles. After about a week a few coleoptiles in each inoculated pot showed yellowish brown areas in different parts. These areas developed into dark brown lesions within a month and some seedlings showed similar lesions on the first leaf sheaths. The general condition of the plants was as good as that of the controls and no plant died. The controls showed no lesions on the coleoptiles. The plants were grown for another month and no difference in vigour could be observed between the controls and the inoculated plants. After this period the plants were cut and the bases of the stems incubated after external disinfection. The fungus was isolated from the inoculated plants . The following table gives the results of the experiment in detail:-

Cereal		1*	2*	3*	4*
Wheat	Control	30	0	30	0
	Inoculated	29	11	29	13
Barley	Control	29	0	29	0
	Inoculated	30	8	30	8
Oats	Control	28	0	28	0
	Inoculated	29	13	28	17

1*. Number of seedlings in each pot.

2. Number of seedlings showing infection.

3. Number of seedlings (in parts) incubated.

4. Number of seedlings yeilding the fungus.

As stated the fungus is not a very serious parasite and in nature appears to be only a secondary invader, after the plants have been weakened by some other agancy. The fungus is carried along with the seed from year to year in the form of spores or mycelium. The mycelium is usually found in the seed coat or the tissues of the palea in the case of barley and oats.

Another type of sclerotium was observed on the wheat grains. These sclerotia are dark blue, almost black, and half submerged in the coats of the seeds. As a rule these sclerotia are not visible in a seed sample even if the individual grains be examined. They become very prominent if the seeds be sown in petri dishes and after cutting the shoots, the grains kept as such for about a week. As many as six sclerotia have been observed on a single seed. Strands of brownish white mycelium grow out from these grains and run along the roots and form sclerotia. These latter sclerotia lie loose in the mycelial felt. Later on conidia of Acremoniella cerealis n.sp. develop on the same mycelium. All cultures made from sclerotia, both from the grain coats and the mycelial felt, ~~gave~~ turned out to be those of A. cerealis, but no sclerotia developed in any of the cultures.

Cultures of Acremoniella were made on wheat grains but no sclerotia of any description were obtained. No other type of spores except those of Acremoniella were formed in culture.

It appears that either the fungus forming sclerotia is different from Acremoniella and is a sterile form or it is the same fungus (Acremoniella) which loses its power to form sclerotia when grown in culture. This point could not be settled as it is impossible to keep the grains sterile for a long time in petri dishes, and all sorts of contaminations exclude the possibilities of getting a pure culture from these sclerotia. I am of opinion that the fungus forming these sclerotia is absolutely different from A. cerealis.

Since the fungus could not be grown in culture in a pure form no infection experiments were carried out with it. Naturally infected grains were sown and compared with uninfected grains, and it was found that the infected grains formed very poor seedlings. Some times Fusarium sp. was found along with these sclerotia, but this species also failed to form sclerotia in pure culture.

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Variability.

That the phenomenon of variability is exhibited by the members of the genus Fusarium and some other organisms is a fact universely recognised and many writers have attempted to explain the causes and nature of this variability.Some writers are of opinion that this change or variation in any organism is a definite transformation of the fundamentals of the characters of the fungus, and they apply the term mutation to all such visible variations. Others beleive that the change is merely a reaction of the organism towards its environment and that the phenotypic change is not accompanied by a similar change in the genotype. Others again are of opinion that all the characters exhibited by any organism under any conditions are a revelation of the true nature and capabilities of the fungus and that there is no reason to beleive in the existence of any such mysterious phenomenon as variability of the nature of the organism. All characters exhibited by any organism remain within the species orbit and that the display of any new characters,unknown to the species before,should be regarded as an addition to our knowledge of the capabilities and nature of the species and nothing more.

Whatever attitude one may adopt, it is an undenia able fact that variations in phenotype of the fungus do occur and that in many cases such variations,so far as we know, are permanent. In other cases the variations are temporary and /

and these temporary changes are either reversible or non-reversible. As a rule temporary reversible changes are exhibited as a~~/~~ reaction towards the environment under which the organism is grown and the fungus reverts back to the normal type as soon as the factor responsible for the change is removed and the organism grown under normal conditions. Sometimes it takes two or three generations before the fungus reverts to the normal type.

Before we begin to discuss these changes in detail certain facts must be borne in mind. The first is the recognition of the 'norm' or 'type' growth of the fungus species in question. Commonly when a fungus is grown in culture on media like oat agar at a fixed temperature a certain type of growth with some definite characters is obtained. This growth need not be normal which I call here 'norm', but for the study of the cultural characters this growth may be taken as the'type' growth and when comparing any subsequent variant with the parent, the two should be grown under the same conditions which produce this 'type'. The cultures should be grown in triplicate and the experiment be repeated at least twice. If under these conditions the variant still differs from the parent, it should be regarded as a variant, but only with reserve. It should be compared with the parent again and again for a long time and if the difference still persists the variant should be taken as a definite permanent variant. All organisms when compared should be grown on the /

the same medium and before the final comparision is made it must be ascertained that all the organisms that are being compared have been growing for atleast three generations under similar conditions and that similar inoculums were used in making cultures. In this way we recognise different variants and the magnitude of the difference depends upon the nature of the change. There are differences in cultural characters, physiological characters and so on, and with the aid of these characters we recognise the physiologic forms, races, biotypes and varieties. Under the cultural characters are considered the nature of the growth, the colour of the mycelium, power to form certain types of spores and so on. Under the physiological characters are considered the temperature requirements of the fungus and its ability to grow at various Ph. The physiologic forms are distinguished from each other by differences in cultural characters and physiological characters. The races are distinguished from their comparative pathogenicity and differences in cultural characters. The biotypes are distinguished from differences in parasitic qualities, as to say one biotype may be parasitic and the other saprophytic. The varieties are distinguished from differences in morphology. There is always a difference in the cultural characters of different varities.

If the above instructions are followed in a study of the genus Fusarium, one will come across a few types of variations in these fungi, and it will be noticed that many so-called variations /

variations are merely changes in the phenotype due to differences in nutrition given to the fungus and the temperature at which the cultures are grown. These changes are very remarkable in certain cases, but they are easily understood as adoptive variations. When the fungus is grown under the normal conditions for the production of 'type', it will revert back to its original form almost immediately within one or two generations. A typical example may be given here from the text. F.28 (a), when grown on oat agar, does not form any aerial mycelium and forms abundant orange sporodochia which sometimes appear as pionnotes. The same fungus when grown on potato-dextrose agar forms abundant aerial mycelium and few hazel sporodochia. This change is a reversible adoptive change and can be produced at will. Another example of such a variation is the change of colour from reddish to yellowish or vice versa when the same fungus is grown at different Ph.

Another type of change results from the type of inoculum used for making cultures. Cultures made from sporodochia tend to produce abundant sporodochia, but little aerial mycelium, and if this practice be repeated the production of aerial mycelium may be considerably reduced. If the cultures be made from the aerial mycelium, the production of the colour in the medium and the formation of sporodochia is greatly reduced, so much so that no sporodochia develop after a few generations if the cultures be repeatedly made from the aerial mycelium. If, in the former case the cultures are made from the scant aerial mycelium this type of mycelium will again become /

become abundant and the cultures revert to the original 'type'. In the latter case the 'type' is obtained if the cultures are made from the spores that occasionally develop in the aerial mycelium. This type of change I call temporary selective variation and this change can also be produced at will.

There are other variations similar to those already described, but these changes do not affect the organism materially and the nature of the fungus remains unchanged. It is not these types of variations with which we are concerned. The real difficulty comes in when the organism changes its phenotype and differs from the parent in certain characters which so far as we can ascertain remain fixed. Commonly such variants arise as sectors in a rapidly growing colony, but sometimes they may also appear as bounded areas in a slow growing colony. The variant either differs from the parent in colour or the presence or absence of aerial mycelium or some such easily recognisable character. Similar variations also occur in other characters, and in many cases they are overlooked simply because we have not got sufficient means to recognise certain differences in the phenotype and genotype of the fungi.

The recognised variants remain fixed when transferred separately, in many cases and we cannot account for their existance or appearance in terms of adoptive or temporary selective variations. We cannot produce such variants at will. Naturally the question arises what is responsible for the appearance of

of these variants, if change in environment is not the cause of their production? To give arguments in support of the fact that environment and culture media are not responsible for such variations that appear as sectors in colonies is merely a waste of time. A worker on Fusarium realises this fact himself as soon as he gives sufficient thought to this problem, and for those who are not in touch with Fusarium the works of Leonian and others will make the point clear. These preliminary discussions have been given by these authors and the aim of the present discussion is merely to take up the line of argument from the point where it stands at present.

Leonian in his latest work on the variability of Fusarium moniliforme has discussed certain explanations put forward by various workers to account for the existance of variability, and amongst the various causes of variability he has discussed environmental stimuli, contamination, degeneration, hybridisation, mutation and mixo-chimera. He applies the name dissociant to a variant, and I consider this name to be inappropriate. The very term dissociation from which the word is derived means the termination of a union or association. Since we assume the previous existance of combination and complexity in the organism, it is needless to wonder over or reason the appearance of an organism from the complex parent. The only explanation is that the association terminates and there is no mystery about variability. But the variations,as we know, are not dissociations and so the term /

term dissociant cannot be applicable to a variant. In the present discussion the term variant or saltant will be used instead of dissociant.

Leonian rightly discards environmental stimuli as an explanation of variability. This has been discussed before and need not be repeated again. The next explanation is that where by contaminations are held to be responsible for variations. This explanation is the least valuable of all, as we get variations in single-spore cultures and as many as 100 variants have been obtained by certain workers from a single spore. When ever there is a mixture of any two fungi the components sector out and remain free and pure in subsequent generations. Some people beleive that even single-spore cultures are not necessarily pure and that a single spore may contain more than one nucleus and these nuclei may come from different organisms. If this be accepted then what is there to stop us from beleiving that different chromosomes constituting a nucleus come from different organisms. Then again why should a nucleus be considered more important than the cytoplasm and why should it be supposed that the nucleus carries the characters of the organism and why not cytoplasm. This question will be discussed in my own explanation of the causes of variability.

Degeneration and Hybridisation are out of question in the case of Fusarium species as we cannot suppose the presence of such phenomena without any proof of their existance in these fungi.

The next explanation of the causes of variability is that given by Brierly and termed by him mixo-chimera. According to this hypothesis somatic fusions between the hyphae of different organisms may give rise to new forms. It is beleived by the exponent of this theory that such somatic fusions are quite common and that this phenomenon is perfectly normal in biology. It is perfectly true that somatic fusions do occur and there is a definite fusion of cell walls and mixing of the protoplasm, but this only occurs naturally when the fusion is between the hyphae or spores of the ~~a~~ same~~o~~ organism and such fusions do not affect the nature of the fungus. Fusions between the hyphae of different organisms can be induced artificially, but the resultant cell formed by the fusion of different cells produces only one type of organism (only one parent), the other being left behind in the process of development. Needless to say new forms cannot arise in this manner and so mixo-chimeras certainly do not explain our point.

Leonian is of opinion that the existence of dissociations in an organism is just the exhibition of the potentialities of the organism and that each organism in doing so repeats the variability of the species to which it belongs. According to him it should be regarded as a highly normal and natural behaviour, and that all species are more or less variable, and they should all dissociate if given the right environment. If this supposition be true there can never be a variation in any species, and if there happens to be any, it can /

can be explained by assuming that the new character already existed in a potential form in the protoplasm of the organism and that the display of a new characters is merely an addition to our knowledge of the potentialities of the species. In this manner it is impossible to distinguish between any two species, and for the apparent fallacy of this argument his own statement which he makes in the final paragraph of his paper on the variability of Fusarium moniliforme speaks for itself. He does not explain any where in his paper why the dissociation occurs apart from his supposition that the power to dissociate already existed in the organism. It appears to me that there are no grounds for such a supposition and to adopt such a passive attitude towards such an active phenomenon is extremely dangerous in the interests of our knowledge of the nature of fungi or any other living organism.

The aim of a scientist is not merely to explore the various avenues of nature, and wonder with awe at the complexities and intricacies exhibited therein. We seek to understand and admire the mysteries of nature not through theoratical philosophy but experimental science. It is true that for the explanation of facts one has to dwell upon ideas apparently irrelevant, but fortunately true and explanatory. With these facts in view and as a student of natural science I shall deliver my opinion upon the subject, and shall only desire it to be esteemed of as little importance as I do myself. By that means you will neither think it worthy of your ridicule nor your anger.

The cause of variability can only be explained if we first understand the nature of the organism exhibiting this phenomenon. Like all other living organisms fungi have a certain type of body commonly known as the thallus. The body consists of certain individual units (cells) linked together in various fashions. The fungi under consideration have a simple form of thallus, consisting of cells linked into fillaments. Each individual cell in itself is an entity and consists of the protoplasm enclosed in a cell wall. Within the cell walls the protoplasm forms a definitely organised physico-chemical system, in which the various constituents act collectively and harmoneously to express certain characters which mark the organism a living matter. Life is merely the outcome of the sum total of the properties of this system, and these properties are the direct outcome of its physico-chemical constitution. The very existance of life and its accompanying properties and activities depends upon the stability and organisation of this system. The nucleus and the cytoplasm collectively carry the characters of the organism, and there is no reason to beleive that the chromosomes alone transmit characters. In cell division the nucleus divides into two equal parts not because it has to distribute the characters equally to the daughter cells, but because the characters of the cell as a whole force it to split in this manner in order to have two similar systems in miniature. It has been definitely proved in Aggregata abernethia that the nucleus does not play any part in the transmission of characters, and /

and cellular differentiation affects solely the cytoplasm. There are such other examples in literature from which the only conclusion that can be drawn is, that it is the cell which guards and transmits the flame of life and its accompanying characters and any thing that affects the cell as a whole will affect its characters. This effect on the cell can only be explained if we ascribe to it a constitution similar to that just described. The physico-chemical system has its environmental limits and when it is affected by ~~its~~ environmental change its stability is affected accordingly. The influence of the factor or factors responsible for the change in the equilibrium of this system varies with the intensity of the effect. If the constitution of the system is changed permanently and the old system is replaced by a new one, we have the appearance of new characters. These new characters may or may not be displayed as a change in the phenotype of the organism concerned. Factors which affect the system in such a manner that its fundamental constitution is not altered produce only a temporary change in the working of the system. Under the influence of such factors there is a suspension of certain activities in favour of the other in order to meet the requirements resultant from the change in conditions and the system again assumes its normal functions as soon as the influencing factor is removed. Permanent change in the constitution of the system leads to the appearance of certain characters and disappearance of others, and such a change should be termed mutation or permenent non- /

non-reversible variation. The reaction of the system towards its environment without any change in its fundamental constitution leads to the appearance or disappearance of certain characters temporarily. If the effect is such that the system exhibits its 'type' characters immediately after the disappearance of the influencing factor, the variation as a result of such a factor the influence of such a factor should be regarded as a temporary reversible change or variation. If, on the other hand the effect of the influencing factor or factors on the system be lasting for an unknown period, but is not in the form of a change in its constitution, the resultant variation should be regarded as a temporary non-reversible variation. These changes differ from mutations only in their relative stability, and their recognition is not quite easy.

If we apply the above explained constitution to the organisms showing variability in one form or another, we can explain the x causes of variability, and the phenomenon no more remains a mystery. The scope of the present work does not permit me to enter into the details of this theory, but it is proposed to expound it further at a later date, and thus its discussion is suspended for the present.

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Concluding Remarks.

In the foregoing pages an attempt has been made to find out the causes of 'Foot Rot' and 'Root Rot' of cereals and in this connection several fungi have been studied. Given proper conditions, almost all the fungi described in the text can cause 'Foot Rot' and in certain cases the disease may be very severe. The two genera Helminthosporium and Fusarium embrace the chief causal organisms, and the other fungi are also of importance, if they be present in large amounts. Only a few fungi have been discussed here and owing to the limited scope of the present work a lot of these organisms could not be studied in detail. Species of Septoria and Alternaria have been isolated regularly from diseased plants, and in infection experiments Septoria sp. caused a serious seedling blight. For the control of the diseases described in this account several experiments have been carried out, but owing to the limited scope of the experiments the results obtained cannot be recommended for use on a large scale.

All the information given in this work is the outcome of original investigations, except in the case of Fusarium where a little help has been taken from the literature in making certain general statements. The description of all the fungi is original, and all the infection experiments described here have been carried out during the course of this /

this work, and the interpretation of their results is strictly original.

Observations in the fields ~~&~~ and experiments in the laboratory lead me to the conclusion that the chief cause of 'Foot Rot' is infection of the cereals by species of Fusarium. The rest of the fungi though capable of producing a similar disease are not very common. Species of Helminthosporium come next in the scale of the cereal pathogens causing 'Foot Rot'.

In the end I wish to express my gratitude and indebtedness to all the members of the staff of the Royal Botanic Garden for their great interest in the work, and constant help in the present investigation. I wish to thank Dr. Malcolm Wilson in particular for his able guidance and encouragement, and helpful criticism of the work, all of which were sorely needed. I also wish to thank Messrs Gartons who supplied almost all the cereal seeds needed in the present work.

THE END.

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Plate. 1.

A B.

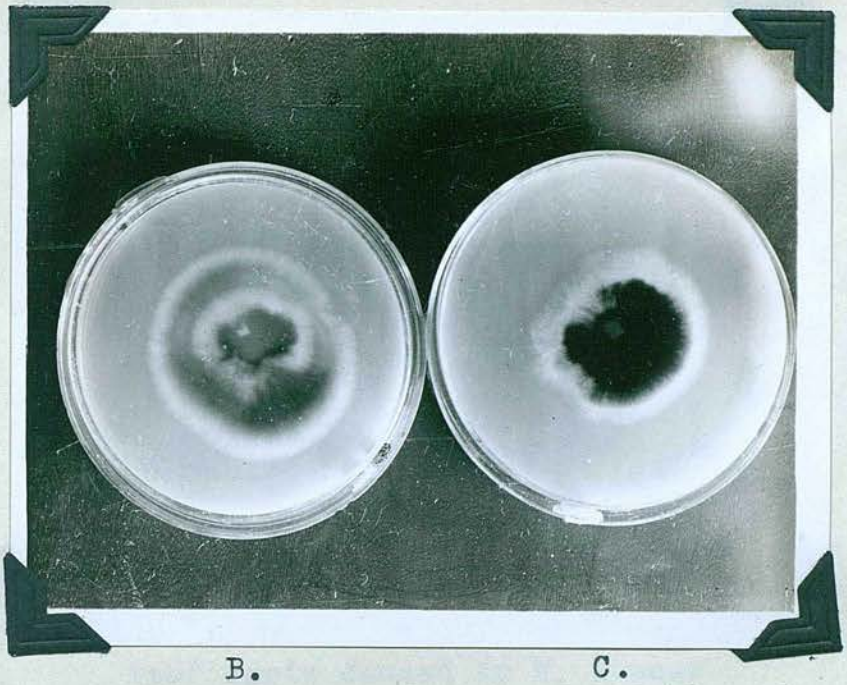
Helminthosporium avenae. Strains A and B. compared on malt agar.

Plate. 2.

A C.

Helminthosporium avenae. Strains A and C compared on malt agar.

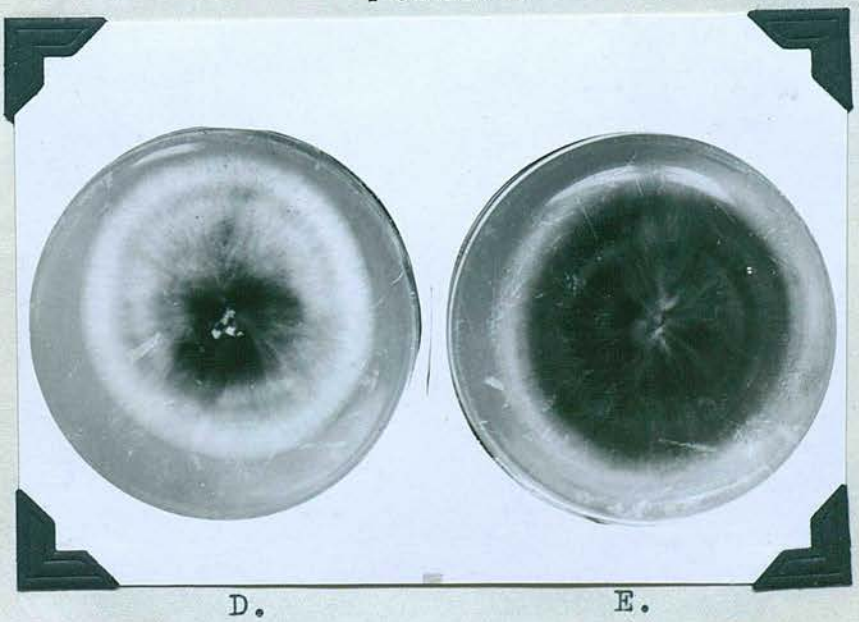
Plate. 3.



B. C.

Helminthosporium avenae. Strains B and C compared on malt agar.

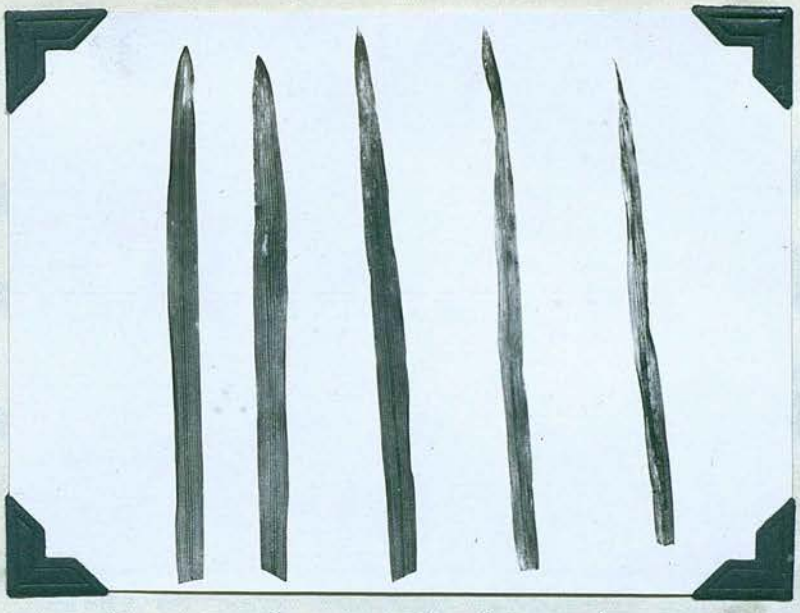
Plate. 4.



D. E.

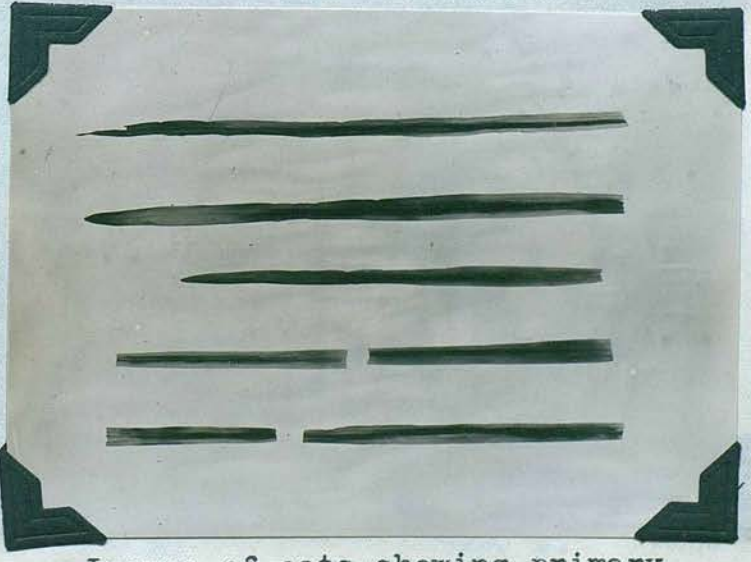
Helminthosporium avenae. Strains D and E compared on malt agar,

Plate. 5.



Leaves of oats showing primary leaf spots caused by H. avenae sativae. (Strain A.).

Plate. 6.

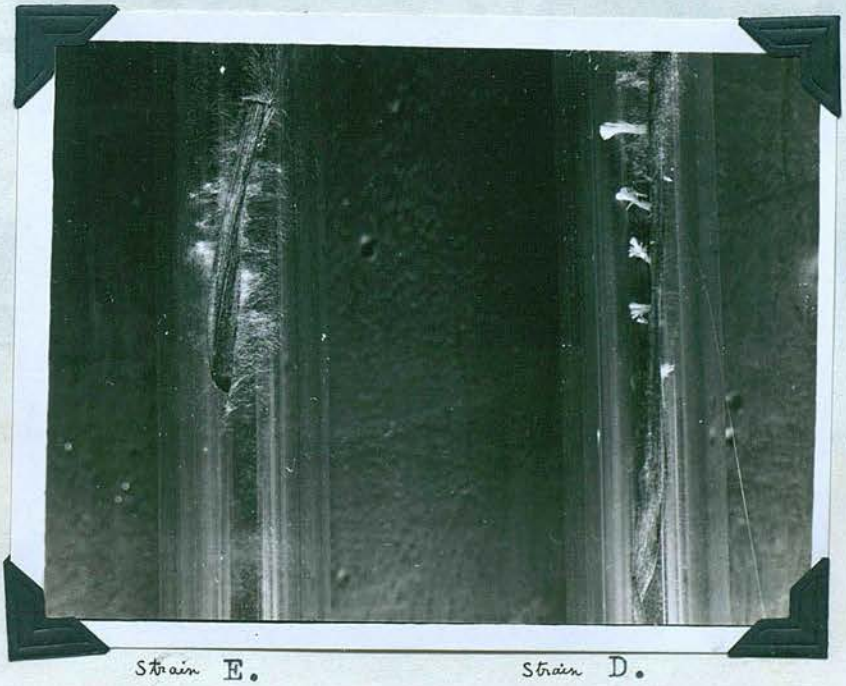


Leaves of oats showing primary leaf stripes caused by H. avenae graminae (Strain D.)

Plate. 7.

Leaves of oats showing secondary infection spots.

Plate. 8.



Strain E. Strain D.

Mycelium of H. avenae graminae growing out of infected leaves after a week's incubation.

Plate. 9.

Inoculated. Control.

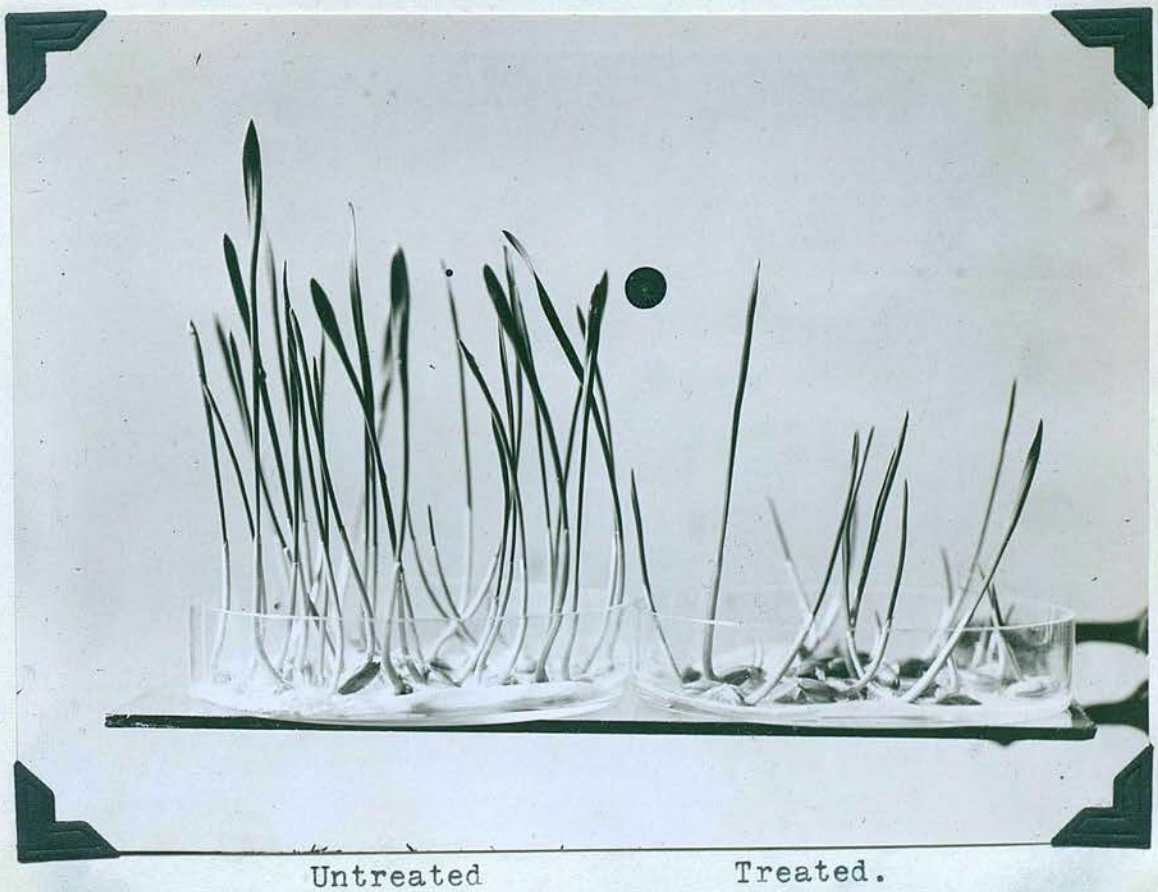
Oat plants infected by H. avenae sativae strain B. Note the serious seedling blight stage.

Plate. 10.

Control. Inoculated.

Oat plants infected by H. avenae graminae, strain D. Note the serious seedling blight stage.

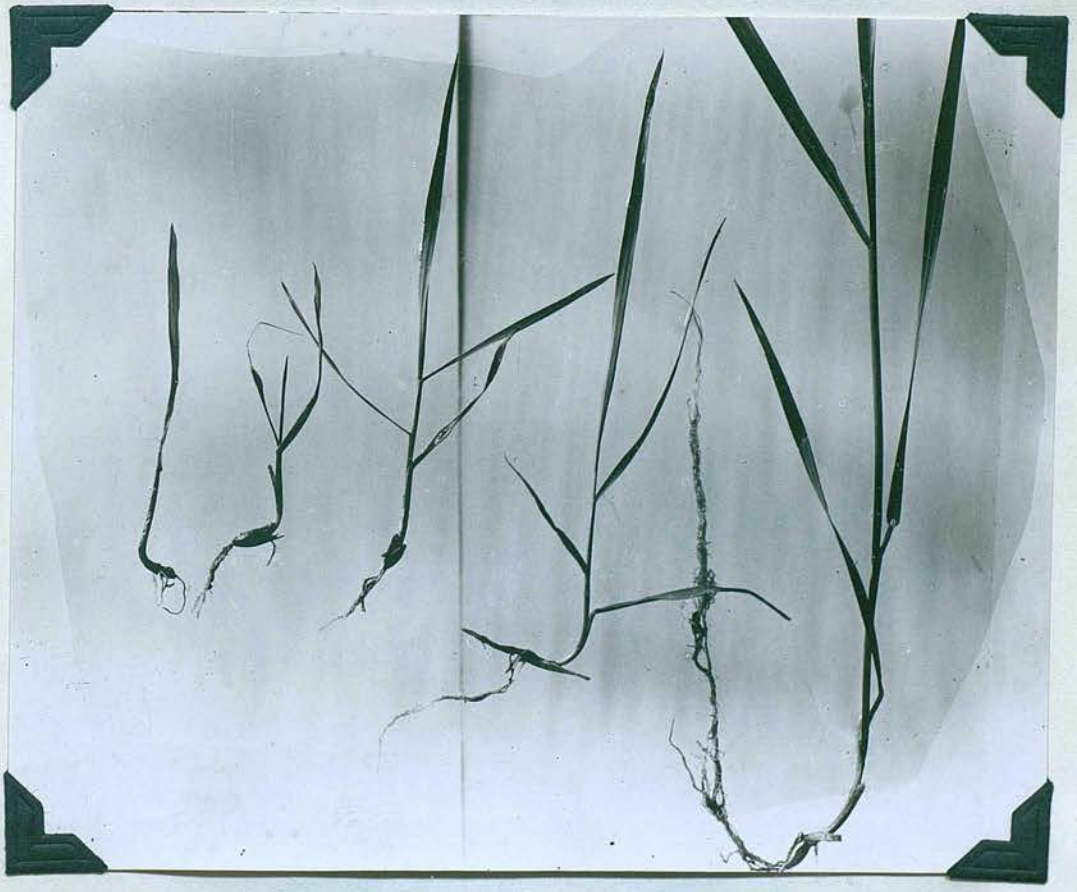
Plate. 11.



Untreated Treated.

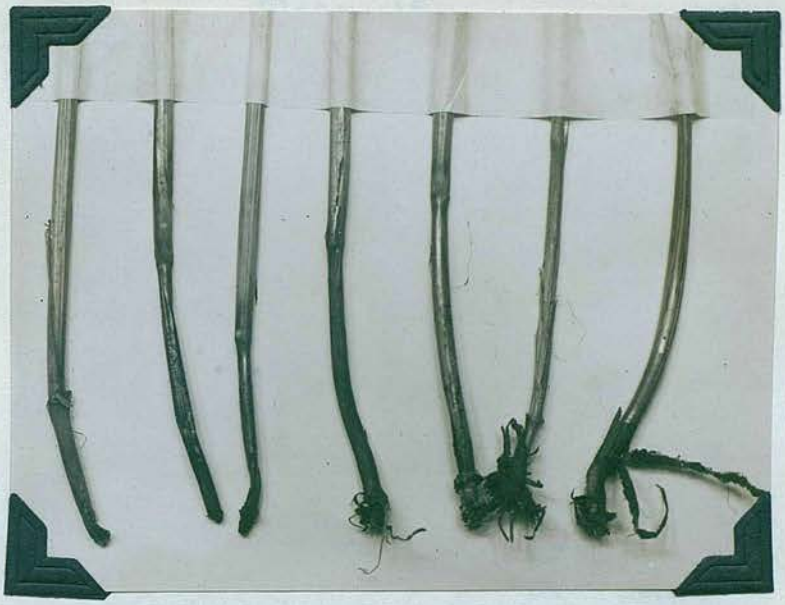
Picture showing the effect of Ceresan on oat plants when grown on filter papers.

Plate. 12.



The effect of Fusarium herbarum /F.avenaceum (F.26) on oat seedlings. On the right hand side is a healthy seedling.

Plate. 13.

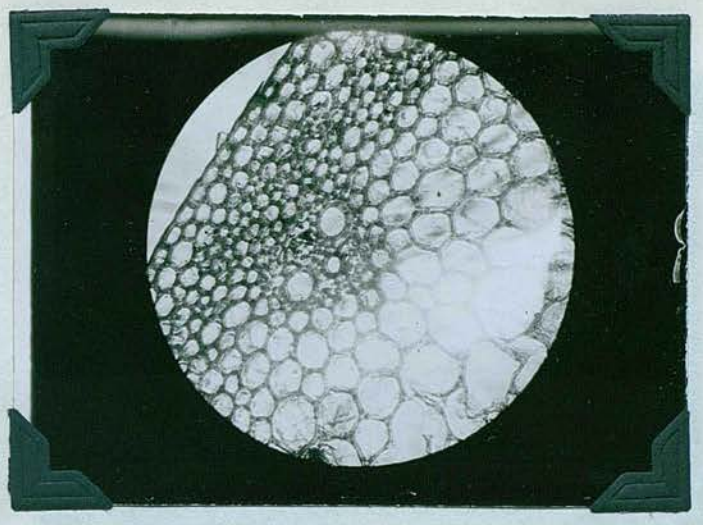


Culms of wheat showing the effect of foot rot. Note the absence of roots in all cases. A secondary root has developed in the case of the plant on the right hand side.

Plate. 14.

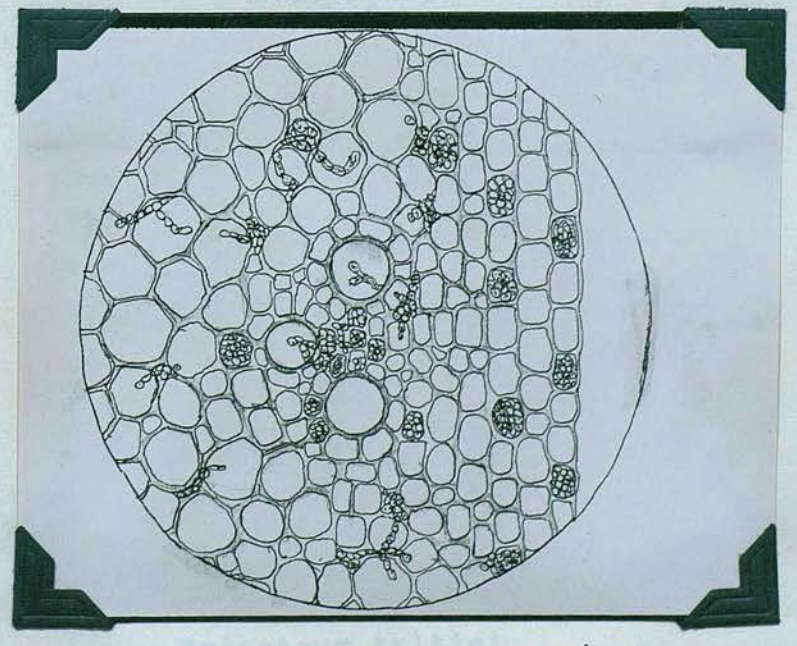
Mycelium of Fusarium growing out of the infected plants and spreading on to the soil.

Plate. 15.



Microphotograph of a section of a stem of wheat infected by Fusarium sp.

Plate. 16.



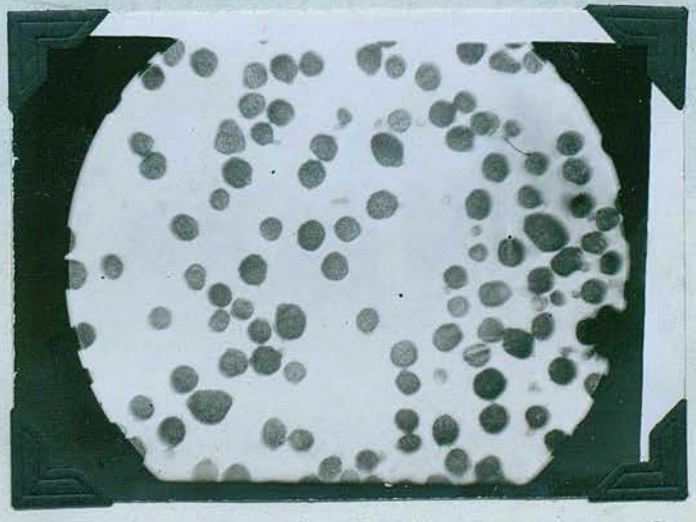
A drawing of the above micro-photograph.

Plate. 17.

Microphotograph showing spores of

Fusarium culmorum.

Plate. 18.



Microphotograph showing spores of

Epicoccum tritici.

Plate. 19.

Inoculated. Control.

Photograph showing the effect of Fusarium on wheat plants.

Plate. 20.

Control. Inoculated.

Photograph showing the effect of infection by Fusarium sp. on barley plants.

Plate. 21.

Control. Inoculated.

Photograph showing the effect of infection by Fusarium on rye plants.

Plate. 22.

Control. Inoculated.

Photograph showing the effect of infection by Fusarium sp. on oat plants.

Plate. 23.

CONTROL.

INOCULATED.

Photograph showing the effect of Fusarium infection on wheat plants.

Plate. 24.

CONTROL.

INOCULATED.

Photograph showing the effect of Fusarium infection on oat plants.

Plate. 25.

Rye Barley

Photograph showing the effect of Fusarium infection on Rye and Barley plants.